Supplement to

THE MEDICAL JOURNAL OF AUSTRALIA

No. 16. SATURDAY, DECEMBER 10, 1927. SYDNEY.

Transactions of the Australasian Medical Congress
(British Medical Association)

Second Session: Dunedin, February 3 to 10, 1927

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COMBINED MEETING.—SECTIONS I AND VIII.

LOCALIZATION OF SPINAL TUMOURS.

By J. F. MACKEDDIE, M.D. (Melbourne),
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(*Continued from page 480.*)

Others have a partially exhausted syringe fitted into the needle so that immediately the *cisterna* is entered, fluid must rush into the barrel. Here, again, it has been found that a fragment may engage in the needle and prevents the barrel filling and under those conditions pushing on may be very dangerous. On the whole I think that Purves-Stewart's method is the best. Take your bearings most accurately, examine from time to time the line of your needle, use your stilette effectively and have a sharp needle. The last is of paramount importance. A blunt short point is more deadly than a very long, very sharp point. One with a moderately short bevel and very sharp is the ideal needle.

One more point. In spite of most careful alignment you may strike bone. A careful survey of your planes will hardly fail to disclose that you are pointing just on the high side and therefore it is occiput you are hitting or on the other hand it is atlas. You must then withdraw your needle a considerable way and make a fresh line of thrust. You cannot "wangle" your way in.

There follow now the reports of some of the cases that have made this article possible; in presenting them I may state that I have refrained from giving detailed neurological findings, contenting myself with those that lent themselves most readily to localization.

CASE I.—Mrs. L., *ætatis* forty-six, for many years a seamstress at the Alfred Hospital, suffered four years ago from numbness in the right thigh and then in the left, succeeded by weakness of both legs. There had never been any pain at any time nor bladder symptoms. Examination at that time revealed only the loss of the right abdominal reflex. Subsequent examinations revealed the classical signs of an advancing spastic paraplegia ending in almost complete paralysis with definite sensory phenomena. No compression was suspected.

The upper limit of sensory disturbance was ill-defined. A pin prick was not recognized as such below the lower border of the second lumbar segment. Between that line and the twelfth dorsal dermatome it was felt as possibly something sharp. This was as nearly as possible alike on both sides. There was no suggestion of a hyperæsthetic area. Limits of effective tactile sensation were much less easily defined. The posterior column conduction was greatly diminishedd.

Only the lower abdominal reflexes were abolished Beavor's sign was not noted. "Lipiodol" injected through the *cisterna magna* was held up at the level of the eighth vertebra (see Figure I.).

Examination after its introduction gave the following results. There was now a band of very great hyperæsthesia running along the tenth to the eleventh dermatomes, while the more or less indefinite delimitation of sensory disturbance became more definite. Mr. McClure, who saw the patient at this stage, put two questions to her: "Have you or had you at any time any pain?" "No." "Have you had any trouble with your water?" "No."

The absence of these classic symptoms of spinal compression greatly discounted the "Lipiodol" findings in his mind and he consented to explore with some misgivings. The eighth and ninth thoracic laminæ were removed and the "Lipiodol" demonstrated. A small, smooth tumour lay on the left latero-posterior aspect of the cord without involving any of the nerve roots. Its removal was a matter of great difficulty and anxiety, for it was apparently incorporated in the substance of the cord. Fears were entertained at the operation that considerable damage was done to the cord. The condition of the patient for some weeks afterwards seemed to confirm our fears, the condition being as nearly as possible that of a total transverse lesion with complete retention of urine and profound anæsthesia and lost deep reflexes. However, there soon appeared signs of improvement and six months after the operation she was walking about with the assistance of somebody's arm and reported that she was getting steadily better.

Mr. Trinca's note on the tumour is to the effect that it had the structure of a typical psammoma.

CASE II.—Nurse R., a graduate of the Alfred Hospital, was seen some four years ago for "nerves." She had severe pain in her back and complained of being "run down." No neurological findings were discovered at that time. Two years later she complained of pain in her back and down the right thigh; there was undoubted rigidity of the lower extremities. Abolition of the left abdominal reflex was the only pathological finding. In the middle of 1925 she manifested great spasticity with ankle and patellar clonus and great difficulty in walking; both abdominal reflexes were absent and there was definite bladder and rectal trouble.

There was no alteration of sensation of any kind anywhere.

Dr. Sewell saw her with me at this stage and in view of the fact that there was some upper limit indicated by the persistent pain that had been a very early symptom, and of the onset of retention symptoms, we felt that "Lipiodol" was indicated even if there was no Froin's syndrome of loculation and an absence of all sensory phenomenon. The result is shown in Figure II., in which the "Lipiodol" is seen resting after an unobstructed course down the theca in the *cul-de-sac*.

She was being investigated at the same time as Mrs. L. and the pair made a very interesting study.

The surgeon's inquiry as to bladder symptoms and pain would have been answered in her case in the affirmative and yet there was no compression.

CASE III.—H.W., *ætatis* forty-six, a salesman, gave a history of twenty months' pain in the back and legs more severe posteriorly. All the varieties of pain were described by him as "stabbing," "dragging," "numbing" and "paralysing." He passed his nights standing at the head of his bed gripping the head rail for support till on his fingers corns actually developed. There were no bladder or rectal symptoms.

Repeated examination revealed a complete absence of any neurological abnormality, either motor or objective sensory or reflex. Lumbar puncture yielded fluid with all the evidence of an undoubted Froin's syndrome, indicating a thecal obstruction. An attempt was therefore made at localization from symptoms alone. There was an area not encroached upon by pain; that was the region corresponding to the distal (that is on the back of the thigh the upper) half of the sacral distribution and the perianal area of the third, fourth and fifth sacral and coccygeal. This exempted the contribution of the *conus* to the *cauda* and thus marked off very definitely the lower limit of the obstruction. Pain in the back was as high as the first lumbar distribution. This, of course, could be caused by compression of the lumbar distribution at its exit from the theca, in its course in the theca, or at its actual separation from the cord itself. The upper limit was much less definite; nevertheless as the "Lipiodol" skiagram shows, a very fair guess at the position was possible. "Lipiodol" injected through the *cisterna magna* came to rest at twelfth thoracic to the first lumbar vertebra (see Figure III.).

Examination after the "Lipiodol" was in the cerebro-spinal fluid made more certain of the painful areas above described, as the pain became intense, paroxysmal and very sharply defined. But no objective neurological phenomena could be demonstrated.

An operation was indicated. The cord was exposed, the "Lipiodol" was retrieved and a small soft hæmorrhagic growth removed. It lay with its lower end just reaching the *conus*, wrapped round by the *cauda* and attached by two large polar vessels to the *pia arachnoid*. It was easily removed leaving the structures intact. Apparently it was too soft to cause compression signs.

For the first time for nearly two years the patient passed a peaceful night in bed and his subsequent recovery was complete.

This patient was referred to me for "Lipiodol" investigation by my colleague, Dr. A. V. M. Anderson, who has kindly given me permission to publish his notes of the following case. The tumour was removed by Mr. Brown.

Mr. Trinca's report is to the effect that the tumour consisted of a number of large blood spaces lined by tumour cells arranged in a diffuse manner without any definite intercellular substance. The vascular element was extensive enough to justify the name angioma. Individual cells in shape and arrangement resembled carcinoma rather than sarcoma. The matrix was represented by a lowly nucleated form of fibrous tissue.

The diagnosis was in consequence angio-endothelioma.

CASE IV.—Mrs. K. suffered from a condition very similar to the preceding patient, H.W. Repeated examinations failed to disclose any objective neurological findings and yet the pains in her back and legs had been more or less continuous and severe for six years. During the last six months the pain had been much intensified so that sleepless nights, even with sedatives, were the rule.

The cerebro-spinal fluid yielded evidence of a typical Froin's syndrome which is almost pathognomonic of obstruction. An attempt to localize the obstruction from symptoms only, as in the last case, was interesting. Unlike the case of H.W. there was no area of sacral distribution free from pain, that is the lowest components of the *cauda* were involved and one would expect the block to be located lower down. Pain reached as high as in H.W., that is at the first lumbar distribution. Therefore, although the same indefiniteness obtains concerning the upper limit, we are enabled to say that the obstructing body is longer and its probable location is at or just below the *conus*.

It is to be observed here that in spite of the fact that the conal component of the *cauda* was involved, there were no bladder or rectal symptoms.

Immediately on the introduction of the "Lipiodol" the upper limit of the painful areas was strikingly picked out by an acute exacerbation of the pain. While this upper painful area was somewhat more sensitive to handling, there was nevertheless no abnormality in the way of anæsthesia below this line or hyperæsthesia above it.

The "Lipiodol" injected through the *cisterna magna* came to rest at the first lumbar vertebra (see Figure IV.).

With the guidance of the "Lipiodol" picture the lamina of the first lumbar vertebra was removed and the "Lipiodol" released. The actual obstruction lay under the lamina below; on its removal a tumour very similar in appearance to that of H.W. was disclosed. It was packed tightly among the components of the *cauda*, its upper limit just reaching the *conus*; it was a little difficult to deliver but was unattached to any cord structures except for two large vessels. It was much longer and stouter than the proceeding tumour, yet in spite of its tight fit in the theca no compression signs manifested themselves at any time.

For the first day or two there were severe pains in both feet, but on the fifth day after the operation the condition was more or less normal and there had been no sign of the old pain, so that a complete recovery is confidently expected.

This patient was also referred to me by Dr. A. V. M. Anderson and again Mr. Brown was the surgeon.

Mr. Trinca, our pathologist, made notes to the effect that the tumour consisted of cells arranged as in the common so-called dural endothelioma; although the tissue was highly vascular, the blood was enclosed in definitely walled vessels, contrasting strongly with that of H.W. In places there was a tendency to concentric disposition of the cells round the vessels suggesting a peritheliomatous nature.

CASE V.—Mrs. T., *ætatis* forty-five years, began seven years ago to notice pain in the right groin and down the front of the thigh, followed soon by a similar condition of the left side; stiffness of her legs supervened, then dragging of her legs and walking only with help and finally complete paralysis with retention of urine. The bladder later emptied itself automatically and incompletely. Examination revealed a condition of spastic paraplegia with the legs in slightly flexed position. On the right side there was complete sensory loss up to the level of second lumbar dermatome and comparative loss as high as the twelfth dorsal. On the left side all forms of sensation were much diminished, pain and the temperature sense alone were lost at the third lumbar segment. The posterior columns were comparatively intact; joint and vibration sense was almost normal.

A Wassermann test applied to the blood serum and the cerebro-spinal fluid did not yield a reaction. "Lipiodol" injected through the *cisterna magna* was held up at the tenth to the eleventh thoracic vertebra (see Figure V.). When the theca was exposed by Mr. Hurley it was found that the *dura mater* and leptomeninges were bound down over more than three centimetres of the cord in the form of a hard ring. An attempt was made to dissect this off, but was only partially successful.

As usually happens there was a deepening of symptoms; for example, complete retention returned and then sensory loss on the left side deepened to the degree of the loss on the right, but at the end of a fortnight the condition was practically what it was before the operation. The woman returned home to the country and the last accounts were of slow but definite improvement. Needless to say no over sanguine hopes are entertained for this patient.

This patient was referred to me for "Lipiodol" investigation by Dr. Sewell to whom I am indebted for the notes and permission to publish the details .

Dr. Mollison reported on the compression tissue that it was non-specific inflammatory products.

CASE VI.—B.E.E., a labourer, *ætatis* thirty-five, was admitted for a condition diagnosed as a meningo-myelitis of doubtful specific origin. There was a history of a some-

what sudden onset six months previously of pains across the abdomen and in the lumbar and sacral regions. Then followed wasting of the lower extremities and accompanying weakness. There was also rectal and bladder incontinence. Examination revealed a distended abdomen with absence of the upper and lower reflexes; all the deep reflexes of the lower extremities were abolished. There was considerable disturbance of effective tactile sensation and of pain and temperature sense, the upper limit of which at this stage was quite indefinite. The posterior column conduction was affected to a much less extent. Beavor's sign was elicited (the umbilicus moved upwards). Just then a rapid change took place in the area involved; the upper limit receded downwards and the lower limit upwards, so that all abdominal signs and symptoms vanished and the rectal and bladder trouble abated. A careful examination at a later date revealed the following sensory loss. On the right side the recognition of a pin prick was dulled from the level of the middle of the third lumbar dermatome, as far as the middle of the first sacral where it became normal again. On the left normal recognition of pin pricks was dulled at the lower limit of the third lumbar, became absolutely lost at the lower limit of the fifth lumbar as far as the beginning of the second sacral, where it became normal. Effective tactile sensation followed similar but much less definite limitations. The cerebro-spinal fluid yielded a definite loculation syndrome. "Lipiodol" was given by the *cisterna magna* route. The level it took up was not compatible with the latest neurological findings; it stayed at the level of the seventh thoracic vertebra (see Figure VI.). But a curious thing happened. The upper limit of the disturbance took up its former position; the abdominal signs and symptoms returned with all the old pain and the patient said he was as bad as he had been at first. But there was a quick subsidence of all abdominal phenomena, for the "Lipiodol" in a day or two found its final resting place at the twelfth thoracic vertebra and so fitted in with the final clinical findings (see Figure VII.).

Mr. Kennedy exposed the cord over the "Lipiodol" and retrieved the latter. There were dense adhesions everywhere which were freed as far as could be from the caudal tissues. From the nature of the onset and history we were not surprised to find no obstructing growth. There followed distinct improvement, but dramatic results in a case of this kind are not to be expected. Needless to say on the finding of a reaction to the Wassermann test vigorous antisyphilitic measures had been taken and it was only when there was a fixed residue and a definite Froin's syndrome that operative measures were considered. The suspended "Lipiodol" decided the matter.

CASE VII.—H., *ætatis* sixty-eight. On admission his right knee jerk was exaggerated; his left knee jerk was absent. His right ankle jerk was absent; his left ankle jerk was exaggerated. There was a distinct sensory loss over the front of the left thigh which was the seat of intense pain and of considerable wasting.

On the right side the wasting was in the posterior aspect of thigh, affecting chiefly the gluteal region. There had been very great pain down back of thigh and leg with occasional recurrence. There had been a history of a "sciatica" of the right leg some twelve months before and as it disappeared to a large extent, a similar condition supervened in the right thigh, but being anterior it was not called "sciatica." The sensory distribution on the front of the left thigh showed it to be confined entirely to femoral distribution. A diagnosis of a peripheral lesion was made which did not call for even a lumbar puncture. But the patient had been sent in by a surgeon for a "Lipiodol" injection and examination.

The result is shown in Figure VIII. The "Lipiodol" meeting with no obstruction has sunk into the little *cul-de-sac* in the theca somewhere in the neighbourhood of the second sacral vertebra.

Two of our later cases were of particular interest.

MRS. W. had increasing weakness of her right leg and to a slight extent of the left with pains in her back for some two years. The weakness and double Babinski sign being an early phenomenon, a diagnosis of disseminated sclerosis had been made. An arm, however, became involved, with great pain and increasing weakness and some spasticity, and she came into hospital for investigation. All signs and symptoms were referable to a cord lesion in the mid-cervical region. Cerebrospinal fluid removed by lumbar puncture gave evidence of a definite Froin's syndrome and yielded a positive Wassermann reaction. There was the history of severe pain in the back at varying levels, so that one considered the possibility of a *pachymeningitis cervicalis hypertrophica*. Antispecific treatment being of no avail and the delimiting of the compression spot not being so precise as such a critical area as the cervical spine demanded, "Lipiodol" was introduced into the *cisterna magna*. It was held up, as you see, at the fifth to sixth cervical level. It was my first cervical arrest of "Lipiodol" and I was not happy about the immediate result. It increased the pain in her right arm, abolished control of the bladder and rectum and rendered her almost completely paraplegic. This with fever continued for two days and then subsided. The Wassermann report of her cisternal fluid was very disconcerting; no reaction was obtained. I have seen one other patient lately in whom the loculated fluid gave a positive Wassermann reaction and the cisternal fluid failed to do so. It would be interesting to know if such a result has been observed by anyone else.

At the operation a small tumour was disclosed pressing laterally on the right side of the cervical cord and pushing the cord to the opposite side of the bony theca. Signs and symptoms referable to a lesion of the posterior column had been conspicuously absent and it was seen at the operation that pressure on the cord was almost directly lateral. The right fifth root was involved in this direct pressure of the tumour. The left lateral tracts of the cord were only moderately under pressure, which explained the patient's surprise at her feeling a pin-prick on her bad leg (right) and not feeling it at all in her good leg—a very definite Brown-Séquard syndrome.

This operation was characterized by the strikingly small laminectomy found necessary by the surgeon, Mr. Trinca, and by the fact that this was the only case in which the "Lipiodol" was not retrieved. During the operation the head was well over the end of the table and presumably the "Lipiodol" found its way up into the *cisterna basilaris*.

In Mrs. F. there had been increasing weakness and heaviness of legs for eighteen months, going on to spasticity and a double Babinski's sign. There were varying sensory phenomena which were referable finally to a lower thoracic distribution. There had been no pain nor bladder symptoms and the cerebro-spinal fluid did not yield a Froin reaction. Nevertheless, recourse to "Lipiodol" was had. It was held up, as you see, at the ninth thoracic vertebra, but the picture of the following day, showing the "Lipiodol" down in its normal resting place, was upsetting.

All the same, operation was proceeded with and the explanation found. There was a central tumour of the cord which apparently by some excess of swelling could block the theca intermittently, but not sufficiently to alter the character of the fluid below the obstruction.

The main lesson to be drawn from the above is that:

(i.) The use of "Lipiodol" reveals compression to be a commoner cause of spinal lesion than was formerly thought,

(ii.) In known cases of compression it is a boon to neurologists and surgeons in localization and operation.

Quite recently a new "Lipiodol" has appeared. It is of lower specific gravity than cerebro-spinal fluid, is injected at the usual site of lumbar puncture, rises fairly rapidly in the theca and finally lodges somewhere in the subarachnoid space in the

cranium—"Lipiodol Ascendant." In the following cases it was used in conjunction with "Lipiodol" by the cisternal route to confirm its value.

A young man had severe and long continued pain over the upper portion of the left thigh, passing into the perineum. There were no distinct objective neurological findings. Froin's syndrome was elicited. Figure X shows the "Lipiodol Ascendant," which had been inserted in the fourth lumbar space, blocked one vertebra higher. To confirm this a "Descendant Lipiodol" injection was given by the *cisterna magna* route; the result is seen in Figure XI. There is a definite space in the theca unoccupied by both the ascendant and descendant "Lipiodol." He is awaiting surgical interference.

In doing the early "Lipiodol Ascendant" injections we first injected some "Lipiodol Gravis" at the lumbar puncture and some minutes later the ascendant, to contrast their diverse movements. This will explain the bleb down in the sacrum in these ascendant cases.

A woman had definite objective neurological findings indicating a caudal lesion, but the exact site was indefinite. Froin's syndrome was present. The "Lipiodol Ascendant" is seen in Figure XII to be blocked almost at the start of its journey.

Figure XIII is interesting. In making the cisternal puncture my Resident Medical Officer, Dr. Travers, successfully punctured the cistern and collected some cerebro-spinal fluid, but did not make very certain that his needle was still in when he delivered the "Lipiodol." The "Lipiodol" is in the tissues of the neck and not in the theca; a very easy blunder to make.

Figure XIV shows the final lodgment of most of the "Lipiodol Ascendant" in a case in which there was no obstruction—probably the *cisterna basilaris*. The passage of the "Lipiodol" into the cranium is unnoticed by the patient and has no after ill effects of any kind.

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THE LOCALIZATION OF SPINAL CORD TUMOURS.

By J. RENFREW WHITE, M.B., B.S. (N.Z.),
Dunedin.

IN this paper I shall deal only with the non-neurological or mechanical and radiological means of investigating the existence and position of tumours compressing the spinal cord, whether these be extramedullary or intramedullary.

EVIDENCE OBTAINED ON LUMBAR PUNCTURE.

The Character of the Cerebro-spinal Fluid.

Xanthochromia.

Xanthochromia, a condition of yellow fluid, was first described by Busch in 1897, the year in which Sir Victor Horsley was the first to remove a tumour, a fibro-myxoma from the upper dorsal region of the cord in a man forty-two years of age. Its occurrence with spontaneous coagulation and an increased cell content was first described by Froin in 1903. The further refinement of testing the fluid for the presence of globulin was introduced later by Nonne.

If the fluid obtained by lumbar puncture in a case of suspected tumour exhibit Froin's sign, in most cases the level of the tumour is above that of the puncture or of the highest puncture giving signs of Froin's characteristics. Cushing and Ayers, however, have shown that in occasional cases the fluid above the level of the tumour may exhibit xanthochromia.

The Pressure of the Fluid.

One of the most certain methods of localizing the level of the tumour if it be causing a partial block in the subarachnoid space, is by the performance of multiple lumbar punctures with the object of demonstrating difference of pressure above and below the tumour. This may be done by directly measuring the pressure above and below by means of manometer, particularly by the use of Queckenstedt's method of artificially raising the pressure by jugular compression. By making punctures at points sufficiently far apart and gradually approximating the levels of successive punctures in cases in which there is evidence of distinct pressure differences in the different manometers, the exact location of the block and hence of the tumour can be determined.

Another method is to count the number of drops that flow from the different needles and compare the rate of flow.

In one of my cases the upper and lower of three needles gave an equal pressure and similar reactions to jugular compression and relaxation. The middle needle failed to register very much in the way of positive pressure at all. Operation showed that this needle had struck a cyst, whilst the other two were in the subarachnoid space which yielded no evidence of block.

The Injection of Substances into the Subarachnoid Space.

Substances may be injected into the subarachnoid space for the purpose of demonstrating a block by radiographic means. In regard to the possible dangers of "Lipiodol" injection, Sharp, of New York, has called attention to the fact that "Lipiodol" at times is not absorbed, but becomes encysted; in one case at any rate it formed a tumour which caused pressure symptoms necessitating an operation for its removal. The writer has an X ray plate of a patient's chest in which it is seen that for a period of three months "Lipiodol" had not been absorbed, but had become stagnant at the bottom of the subarachnoid space in the sacral region.

Dandy's method of pneumography would seem to be free from this objection. A lumbar puncture is performed below the level of the lesion; the fluid is removed and its place taken by air which ascends to the tumour, the lower edge of which is thus outlined on radiography.

EVIDENCE OBTAINED BY ORDINARY RADIOGRAPHY OF THE SPINAL COLUMN.

X ray pictures will reveal exostoses and the lesions of spinal tuberculosis, syphilis and cancer.

An important group of cases is that in which osteo-arthritis of the spine causes a more or less generalized narrowing of the spinal canal with compression spread over a considerable length of the spinal cord.

DR. N. D. ROYLE (Sydney) showed cinema films of several patients suffering from the sequelæ of lethargic encephalitis who had been treated by ramisection. Patients with bradykinesia had obtained increased speed of movement, those with residual spasticity became free in their movements and the power of walking and of balancing was restored.

DR. O. DIETHELM (Sydney) testified to the miraculous improvement in the last patient demonstrated by Dr. Royle. He had been suffering from lethargic encephalitis and the sequelæ had made it impossible for the patient to go up steps unless assisted by two people. A section of the sympathetic rami had not only enabled the patient to walk, but had restored the ability to ascend steps.

DR. R. R. STAWELL (Melbourne) complimented the various speakers on their papers. It was splendid to get clearness substituted for confusion. The difficulty with lethargic encephalitis was how to tell whether the condition was going to progress to chronicity. Were the sequelæ not merely manifestations of the original condition? A smouldering fever surely indicated a smouldering inflammatory process, to which could be ascribed the growing rigidity. This could be detected by the pliancy of the muscles. The investigation of reciprocal movement of the muscles, which meant noting a change in muscle tone, was an important extension of neurological signs and symptoms. He referred to the use of lumbar puncture in treatment. He had seen several patients in an apparently desperate condition who had been restored to good health, though with persistent headache, by one, two or more punctures.

In his opinion Dr. Royle's demonstrations had been one of the most impressive features of Congress. He had no doubt concerning the results of ramisection. He suspected that the failure of other workers to obtain results was to be accounted for by faulty technique in a minority and by the unsuitable choice of case in the majority. The suitable type was that of encephalitis or of injury to the brain with cortical or subcortical involvement.

DR. J. F. MACKEDDIE (Melbourne) referred enthusiastically to the new idea, born in Australia, that tone was under control of the sympathetic system. Royle and Hunter had opened a new neurological era.

DR. S. V. SEWELL (Melbourne) subscribed to the view that the complications of lethargic encephalitis were not sequelæ, but a continuation of the inflammatory process. He had been struck by his experience of a case of a man who had had a typical attack of encephalitis. Lumbar puncture had been performed every six weeks. The cell count of the cerebro-spinal fluid at first 10 to 15 had gradually risen and at the end of two years a typical Parkinsonian syndrome had developed. Believing that this indicated persistence of the infection, he had persisted with sodium salicylate over a long period up to six months and considered that progress was more satisfactory.

He testified to the diagnostic value of "Lipiodol" in spinal tumours. By its means much information had been disclosed in obscure cases in which the patient's symptoms had been the only indication of trouble.

DR. J. RENFREW WHITE asked Dr. MacKeddie whether he had given up all other methods than "Lipiodol" in the localization of tumours and whether he had had any experience of compression by osteo-arthritis simulating a diffused tumour. He had operated on two patients with this condition, the first with good result, the second with not such good result.

DR. H. F. MAUDSLEY congratulated Dr. Royle on his demonstration.

DR. MACKEDDIE in reply stated that he had used the air method with little success. Coughing and deep breathing both led to lack of propulsion of fluid out of the canal. The "Lipiodol" method was undoubtedly the best. It had the further advantage that the resident medical officer felt the onus of making a very thorough preliminary examination. Moreover by "Lipiodol" injection a hyperæsthetic area was marked off and so offered an inducement for exact neurological examination. The ascending "Lipiodol" injection was quite harmless.

DR. J. F. MACKEDDIE (Melbourne) showed his method of teaching neurology by means of cinema films. In the first film the method of puncturing the *cisterna magna* for descending "Lipiodol" injection was illustrated. The second pictured Dr. Gordon Homes demonstrating the reactions in a neurological patient.

COMBINED MEETING.—SECTIONS II, IV AND XII.

OSTEITIS FIBROSA AND OSTEITIS DEFORMANS.

DR. H. R. SEAR read a paper on *osteitis fibrosa* and *osteitis deformans* and illustrated his arguments with a series of skiagrams. This paper will be published in a subsequent issue of THE MEDICAL JOURNAL OF AUSTRALIA.

TUMOURS AND INFLAMMATION OF BONE.[1]

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THE distinction between those reactions of bone that are inflammatory and those that are neoplastic, is often difficult to make and if the lesion is a new growth, the degree of malignancy is sometimes not easy to determine. Decisions on these points are of great importance for on them depends the treatment to be followed in each case, sometimes whether a limb is to be amputated or a life risked.

In this demonstration I deal not with the problem as a whole, but with one small part of it, namely the relation, if any, that exists between central giant cell tumours of bone (myeloid sarcomata), simple cysts of bone and localized forms of *osteitis fibrosa*.

My views are not yet clearly focussed, but my provisional opinions are these:

1. Simple bone cysts and central giant cell tumours of bone are akin, for in many simple cysts soft pieces of tissue with the structure of giant cell tumours are present and in some myeloid or giant cell tumours there are portions with a structure resembling that of simple cysts; further some central lesions of bone appear to fall midway between simple cysts and central giant cell tumours.

2. Myeloid tumours sometimes show great variety of structure; fibroblastic tissue, giant-cell tissue, myxomatous tissue and other varieties may be present in different proportions and determine the naked eye appearance of the tumour and its life history.

3. Simple bone cysts do not result from myxomatous degeneration in a giant cell tumour; the formation of myxomatous tissue is to be looked on as an independent tendency of this portion of the tumour to develope along different lines.

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[1] A full description of the lantern slides was given by Dr. A. H. Tebbutt who demonstrated them for Dr. Keith Inglis.

4. Myxomatous softening of vessel walls and liquefaction of myxomatous tissue in and apart from vessel walls are conditions which predispose to cyst formation in bone.

5. After curetting away the soft tissue of a myeloid tumour repair may be so good that skiagrams will reveal no difference between the new and the old bone.

6. A cystic myeloid tumour in a boy showed no tendency to become separated from the epiphyseal cartilage during the twenty-two months he was under observation.

7. Giant cell tumours of bone are new growths, usually of low malignancy. It is exceptional for metastases to occur though occasionally they do so. If the tumour is surrounded by an intact bone shell, curetting is usually the treatment indicated.

8. Histologically it is difficult to draw a line of distinction between a white myeloma in which fibroblastic tissue predominates, and localized *osteitis fibrosa*.

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4. Sir James Paget: "Lectures in Surgical Pathology," Fourth Edition. Longmans, Green and Co., London, 1876, page 547.

5. R. B. Wade and K. Inglis: "Cyst of the Astragalus." THE MEDICAL JOURNAL OF AUSTRALIA, September 11, 1927, page 345.

OSTEITIS FIBROSA AND CYSTICA, OSTEITIS DEFORMANS AND TUMOURS OF BONE.

By F. GORDON BELL, M.D., Ch.B. (Edinburgh), F.R.C.S. (England).

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No subject till recent years has appeared more complex and confused than many of the chronic diseases of bone, because in the main they have been considered as separate entities.

Following Recklinghausen's lead in 1910, however, there have been numerous attempts of late to clarify and correlate many of these conditions such as Recklinghausen's and Paget's diseases, *leontiasis ossea*, otosclerosis, osteomalacia, rickets and even certain tumours and to place them on a common pathological basis.

Taking, for example, Recklinghausen's and Paget's diseases, the many remarkable points of similarity indicate a similar origin though at different age periods, but I feel sure from clinical observation that the age difference has been too much stressed and that it is not so absolute as often stated.

I will indicate in the following table the main general features of these two diseases and will elaborate them as far as time allows.

Pathogenesis.

I shall now sketch briefly the main theories of origin as concern both Recklinghausen's and Paget's diseases.

Inflammatory Theory.

Paget regarded *osteitis deformans* as certainly inflammatory in nature and many subsequent writers have followed his lead. Recklinghausen, too, for many years regarded *osteitis fibrosa* in the same light, but sixteen years ago modified his views and adopted a metaplastic basis in the endeavour to bring a large group of bone diseases ranging from *osteomalacia* to myeloid sarcoma (so-called) into line. If we interpret the term inflammatory in a wide sense and take it as indicating the production in varying magnitude of new fibrous tissue of vascular type, perhaps as the reaction to some chronic toxic influence, then the term would still appear applicable to both conditions.

Lawford Knaggs in a recent series of papers supports the toxic view and considers that the toxins may be metabolic or intestinal in origin. He formulated a working theory to account for the difference in age onset and believed that bone stability is due to some inherent factor which in susceptible individuals may be upset by alleged toxic influences. If resistance is broken down in youth, the severe disease *osteitis fibrosa* results, while in other cases this inherent resistance suffices to stave off *osteitis fibrosa* in youth, but yields to *osteitis deformans* in later life. Knaggs suggests such a view may account for the occasional familial type.

As supporting the inflammatory theory, it may be noted that *leontiasis ossea* and *otosclerosis* which have many points of resemblance to Reckling-

Groups.	*Osteitis Fibrosa.*	*Osteitis Deformans.*
Age	Childhood and young adult life.	Middle and advanced life.
Frequency	Relatively common.	Rare.
Pathological types	(*a*) Localized, fairly common. (*b*) Generalized.	(*a*) Localized: rare. (*b*) Generalized: typical picture.
Bones affected	Humerus, femur, tibia, spine, skull, clavicle, pelvis.	Femur, tibia, skull, spine, bones of upper limb rare.
Clinical types	Pathological fracture common, especially local type. Humerus usually involved.	Pathological fracture rare.
	Bending and enlargement; deformity, especially *coxa vara*. and spine.	Bending and enlargement of femur, tibia, spine and skull.
	Pain, vague rheumatism, growing pains, headache.	Pain.
Course	Progressive (?) 5% spontaneous cure.	Consolidation and restoration of stability; progressive, especially of skull.
Termination	Tumour rare. (?)	Tumour common. (?)

hausen's and Paget's diseases, are probably inflammatory in origin, in some cases at least.

Metaplastic Malacia Theory.

Following Recklinghausen, the metaplastic malacia hypothesis has gained ground. According to it the fibrous tissue substitution, the essential feature in both diseases in the early stages, is regarded as a metaplasia brought about by metabolic influences, the fibrous tissue being subject to a further reversion to bony tissue of a new architectural type.

The causes at work are likely to be varied, for example, faulty metabolism, upsetting calcium stability, overexertion and tissue-ageing as evidenced by the atheromatous arteries of Paget's disease.

Recently the endocrine system has come under suspicion and, I think, with good reason. We know that thyreoid deficiency stunts bony overgrowth and that the pituitary may excite overgrowth and it seems entirely reasonable to suppose that calcium metabolism as it affects bones is controlled by a hormone of endocrine origin, probably parathyreoidal or thyreoidal.

The most convincing case supporting this idea was reported and discussed in all its bearings in 1923 by Dawson and Struthers, of Edinburgh; we have had here recently an almost identical case with a hyperplastic thyreoid tumour in place of a parathyreoid as in the Edinburgh case. The hyperplasia, be it thyreoidal or parathyreoidal, is regarded not as the cause of the bony changes, but as a response to a call for a greater supply of the necessary hormone.

Traumatic Theory.

The trauma hypothesis mainly concerns *osteitis fibrosa* and has a certain measure of support, especially from the French. In a strict sense it is applicable only to the localized type, but with a more generous interpretation may be partly extended to the generalized. It is suggested that the frequent occurrence of *osteitis fibrosa* in the upper ends of the humerus and femur is related to repeated strain of the vulnerable metaphyseal bone of the growing person by the powerful joint movements, just as the metaphyses are so vulnerable to the common infections and to various types of tumour.

Pathological Changes.

The pathological changes consist essentially in a decalcification of existing bone and a vascular fibrous tissue substitution of the marrow and bone to a greater or lesser extent.

Subsequent reconstruction of bone on a new plan occurs in both diseases, to a remarkable extent in *osteitis deformans* and to a variable extent in *osteitis fibrosa*. Cysts and tumour formation of the myeloid sarcoma type are common in *osteitis fibrosa* and probably unknown in Paget's disease.[1]

Treatment.

Localized types appear to respond well to curetting and occasional bone grafting. Dr. Fenwick informs me that violet ray therapy leads to greatly increased calcium retaining powers in bone and possibly this may open up a useful line of treatment in *osteitis fibrosa*, especially in view of the known effect of lack of sunshine in producing rickets and osteomalacia.

Tumours of Bone.

Myeloid Sarcoma.

I propose to consider myeloid sarcoma particularly as it appears to form a link between Recklinghausen's disease and frank tumours of bone. There is a striking resemblance between myeloid sarcoma so called and the localized form of *osteitis fibrosa* and histologically and to a lesser extent on the clinical and X ray side the two conditions are hardly distinguishable. A patch of giant-celled tissue from a bone affected with *osteitis fibrosa* may exactly resemble the characteristic giant-celled structure of myeloid sarcoma.

It is now generally accepted that myeloid sarcoma, better called by Ewing's term benign giant-cell tumour of bone, is in the great majority of cases benign and only locally obstructive and should, where possible, be treated on conservative lines as Bloodgood has convincingly demonstrated.

None-the-less, the giant-celled tumour does occasionally produce metastasis as one of our specimens in the Congress Museum shows.

This tumour, with its relative benignity and giant-celled structure has led to much confusion in the statistics of bone tumours, as it has been confused with those highly malignant growths which may also display giant cells, though of a different type. Furthermore, many successful amputations for the benign tumour are included with sarcoma generally and confuse end results.

Malignant Tumours.

Turning finally to the genuine malignant tumours, I have time to raise only one or two points. In the first place I am convinced that trauma frequently plays the dominant part in the causation of the malignant tumour of bone and I am glad to have the support of so wise and experienced a surgeon as Rutherford Morison in this view. In the second place as regards treatment of malignant tumours I would raise the question of local removal *versus* amputation. It seems to me that the fibula is the only long bone which lends itself to local removal together with the attached muscles and I have personally observed only one successful local removal of an active periosteal sarcoma (lower end of humerus) done by my late chief, Alexis Thomson; the patient was well twelve years later.

In general, the results either of amputation or local removal of active growths are gloomy in the extreme.

Dr. D. F. Myers (Wellington) showed skiagrams of twelve patients with Paget's disease and two with *osteitis fibrosa cystica*. He pointed out that osteosclerosis was usually greater than osteoporosis in Paget's disease.

The first case was that of a man, aged forty, in whom a giant-celled tumour was present in the upper part of the left humerus. Although the arm had been amputated,

[1] The essential changes were demonstrated by lantern slides.

the man had died later from metastases in the lung, clearly visible in the skiagram and of typical snowball appearance.

In the second case there was definite bone absorption conducive to pathological fracture. Out of the series of twelve there had been seven fractures. In this case there was evident osteosclerosis of the skull in contrast with the other bones.

The condition of *osteitis fibrosa cystica* in a child was analogous to Paget's disease in that there were deformity and osteoporosis of the long bones in each, but the skull and spine were never affected in the former. The bones were liable to fracture. Compression fractures of vertebræ did not occur as they did in Paget's disease. In one of the cases shown the malacia had developed as the parents refused to allow the child to rest. Partial fracture of the femur could be seen commencing. There was no family history of disease in either patient.

The next skiagrams were of Perthes's disease in three sisters, aged ten, twelve and fourteen. The one aged twelve had come to hospital complaining of a lump of four years' duration. Changes in both acetabula and in the head of the femur were detected in the skiagrams. The girl aged ten manifested thickening of the femoral neck with acetabular changes and fragmentation of the femoral head. The third sister had had a lump for five years and the skiagraphic appearance of Perthes's disease which had gone on to almost complete healing with good movement at both hip joints was seen.

In discussion DR. P. P. LYNCH (Dunedin) gave particulars of a case of generalized *osteitis fibrosa* in which a central giant celled tumour appeared involving the lower half of the femur. Amputation had been followed by recurrence in the stump. Death occurred from pulmonary metastases with formation of bony trabeculæ in the lung nodules. Histologically the deposit in bone and that in lung were identical; there was no histological difference from the normally benign central giant cell tumour of bone.

PROFESSOR A. M. DRENNAN (Dunedin) called attention to the early forms of bone cysts seen in the case presented by Professor Gordon Bell. He referred to the bone changes in rickets in which from metabolic disturbance of the vitamin A, calcium and phosphorus balance an overgrowth of osteoid tissue occured with deficient calcification. He regarded the changes seen in *osteitis deformans* and *osteitis fibrosa et cystica* as due to endocrine disturbance of the parathyreoids. In the Dunedin case no parathyreoid tissue could be found other than the large nodules of parathyreoid structure, but so far oxyphile cells had not been demonstrated therein. Professor Drennan regarded the myeloid sarcoma or myeloma as a more cellular formation and a true tumour as contrasted with the hæmorrhagic cysts seen in these forms of osteitis which certainly contained large multinucleated cells, but in which the stroma was denser and composed of fibroblasts and osteoblasts.

DR. A. C. B. BIGGS (Balclutha) asked whether in spontaneous fracture of a long bone due to cyst formation operation should be carried out first and the fracture treated later.

DR. MYERS replied that in one patient the fracture had remained ununited for two years, but that in others it had healed quickly.

DR. A. H. TEBBUTT (Sydney) said that it was generally agreed that the term myeloid sarcoma should be replaced by a less misleading term; Ewing's term "benign giant cell tumour" was preferable. Dawson's explanation of *osteitis fibrosa* was very interesting, that the bone cell controlled calcium metabolism in its immediate neighbourhood, that a parathyreoid hormone influenced the bone cell, but certain toxic influences might depress the bone cell activities leading to the calcium absorption; the parathyreoids might be stimulated and might manifest hyperplasia or as in Dawson's case adenomatous proliferations of aberrant parathyreoid tissue might occur. He had been shown an almost identical case by Professor Drennan and having seen a section, thought that there was a parathyreoid adenoma, though a very few colloid containing alveoli were seen. He regretted very much the absence of Dr. Keith Inglis who had taken considerable interest in bone pathology.

DR. W. R. C. STOWE (Napier) cited two cases of Paget's disease that had come under his notice which deserved mention in that they were associated with intense physical and mental activity. Both were in women about fifty years old; one had borne many children; the other was nulliparous. The past histories were free from any conditions of a devitalizing nature. They had led extremely active lives. In the latter the output of energy had been exceptional. Until this dycrasia manifested itself, there was no evidence of physical failure. The assumption, no new one, was that biological fatigue affecting calcium metabolism was a causal factor in Paget's disease.

SECTION III.—OBSTETRICS AND GYNÆCOLOGY.

PUERPERAL SEPSIS.

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IN this paper I shall deal with the problem of sepsis from the standpoint of the general practitioner. Had Dr. White been able to present his paper today we should have had valuable information from the point of view of a research worker. But perhaps it is well that I have to substitute this paper, for just as the problem of sepsis is largely the problem of the general practitioner, much of the remedy may yet rest with us.

Whether we live in the heart of Australia or the farthest north of New Zealand, we practitioners can keep Sir James Mackenzie's maxims before us and remind ourselves from day to day that the only limit to our good practical work and clinical obstetrical research is the limit of our own will and perseverance

I shall not discuss the epidemic type of sepsis; it is no longer a mystery or a problem. From the days when Semelweiss, Oliver Wendell Holmes and other contemporary workers pointed out its infectious nature, the risk of introducing it from the dead house and the risk of carrying it from septic surgical cases, the incidence of this scourge has steadily and creditably declined. If epidemic sepsis (and by this I mean two, three or more of these patients within the walls of one hospital or the practice of one doctor or nurse) was the only type encountered, today there would be no call for a discussion at this Congress on puerperal sepsis, for however widely we may differ in other obstetrical views, we must all agree that the ætiology of epidemic sepsis is ignorance or carelessness and the remedy is nothing short of "eternal vigilance."

I believe that in Australia and New Zealand it is mainly the isolated cases of septicæmia or localized infection with less fever that keep up our mortality and morbidity rates. I do not imply that we do not get epidemic outbreaks now and again, but in the majority of instances there is no history of other persons affected and no grounds for assuming there has been negligence or carelessness on the part of the attendants. Speaking for the New Zealanders, I can assert that this type of sepsis has become the nightmare of the obstetricians and the most dreaded complication of labour. Anything we

can understand, is not so bad, but sporadic fever baffles our reasoning powers by occurring occasionally in our unhandled patients and failing to appear in our difficult, bruised and apparently contaminated patients. The failure to explain envelopes the problem in terror; we can remove an interval appendix with reasonable certainty of result, but by the same application of Listerian principles we cannot pick up an infant emerging spontaneously from its mother and be reasonably sure this normal process will not be complicated by fever.

Current views on the ætiology of this type of sepsis have recently undergone radical changes. The changing outlook was apparent at the British Obstetrical Congress which met in London in 1925 and much has been written on the matter since. Until three or four years ago it was common belief that one law of infection governed both the epidemic and the sporadic type of case. This calm acceptance of Semelweiss's explanation as the final word, the grasping at half truth, in mistake for whole truth, has been nothing less than a scientific and national disaster. Many valuable lives have been lost or impaired, while we have been feverishly working along false clues. As time passed and still the fever rate remained approximately stationary all concerned became more panicked in their actions. Health boards stepped in to suggest to the already distracted practitioners ways and means of preventing exogenous infection; when these ways and means failed, increasingly stringent rules were made, the net result being a spirit of dissension and distrust in a task that called for mutual cooperation and closest harmony. Moreover, we have allowed half truth to go forth to the public as whole truth and commissioners, politicians and suffragettes now add to the general confusion, their main opinions being (in England and New Zealand; I hope Australia is more fortunate) that the doctors are a dirty careless lot, addicted too much to interference and too little to the use of lysol.

The mental torment experienced by thousands of conscientious doctors in the last few decades is the least of many evils. We belong to a profession which is proud to make sacrifices, so we will not waste time in self pity. Far-reaching evil arises out of the fact that we have allowed the public to imbibe erroneous conceptions of the present day type of sepsis.

It is going to take us some years to disperse these views and all this time we shall have public opinion blocking our progress at every turn. I shall speak presently on the question of public opinion, but shall say here that until public confidence in the profession is restored, we shall make no headway. Rightly or wrongly every doctor gets the blame for a rise of temperature, the result being that as far as possible fever tends to be concealed. Many a careful doctor is tempted to allow a patient out of bed on the tenth day, although she has a temperature of 37·4°C. (99·4°F.), rather than risk telling her about it. Moreover, enthusiastic reformers, anxious to bolster up the birth rate, have spread the news that labour is a natural, safe and easy process, with the result the public has no idea that up-to-date midwifery often involves team work for its aseptic conduct and misjudges the man who asks for assistance in difficult cases.

Following on the idea that sepsis was always introduced from external sources, we have preached a doctrine of "non-interference." Some pseudo-authorities have in their zeal preached this doctrine to the public also, with the result that the junior practitioner fears to do the necessary scientific things and fears what the patient will say about him if he does it. Our Australian colleagues may have more moderation both in their injunctions and in the way these injunctions are interpreted, but the slogan in our little Dominion has of late been: "Keep your hands in your pockets and leave things alone," advice which might well be interpreted to read: "Never go to a maternity case till sixty minutes after you are called, for thereby you will escape much work and evade much responsibility."

In more conscientious circles I have heard young doctors say they were never sure whether they were dealing with an occipito-posterior presentation or not, as they had such an obsession of sepsis that they were mortally afraid to make a thorough internal examination. Once they get to this stage their temerity is courting the very thing they are feeling. Internal examinations, when necessary, can be made with perfect safety; it all depends on the man who makes them. Whereas, the man who is afraid of examining, is by unnecessary hours of labour apt to wear out his patient's resistance powers to streptococci, staphylocci and colon bacilli, which are assuredly in her system before he came upon the scene at all. Truly the wholesale adoption of Semelweiss teaching has entangled us in the toils of a vicious circle. The public blames the doctors and the doctors fear the public. The health officials institute rules and the obstetricians try to evade them. Leastwise, we did in this country until a summons or two struck a timely warning, thereafter our returns became more accurate, but at the cost of an ever widening gulf between officials and obstetricians.

The student sees the trials of midwifery and scorning the fee of four or five guineas for such risks, decides to specialize in any branch of medicine other than obstetrics. If later on we find him struggling with obstetrical emergencies for which he never prepared himself, we need not blame the enthusiasm of youth that aspired to gastro-enterostomies instead of breech presentations, rather should we pity him as a man doing obstetrics against his will and the victim of a pernicious chain of circumstances. So unattractive is the subject of midwifery in our country that I think Dr. Riley could tell you he has not had ten students in the last ten years who have expressed any desire to specialize in obstetrics.

If we wish to complete the simile of the vicious circle, we should add that because of the opprobrium of puerperal fever the majority of good practitioners give up this work just at the stage when they have purchased their dearly bought experience and pass on their patients to the inexperienced juniors.

Blank despair might well be our attitude if it were not for a ray of hope that comes in the newer conception of autogenous sources of infection as well as exogenous sources. The newer conception does not promise to absolve us from responsibility; far from it; but it offers us some reasonable explanation. The late Dr. Fothergill and Mr. Bonney have been the chief exponents of this principle, but until recently have been battering their opinions unheeded against a stone wall of conservative opinion, for though text books made mention of this possibility, the ruling authorities held that the doctrine of sepsis lying latent in the patient's own body was too dangerous for promulgation. Fothergill and Bonney have lately received support by research work conducted at the Simpson Memorial Hospital, Edinburgh, the Rotunda, Dublin and St. Thomas's Hospital, London, while Professor Cassidy at the Coombe, Professor McIlroy at the Royal Free and Sir Ewen Maclean in Wales are all working on the parallel question of resistance.

In Edinburgh they examined one hundred intrauterine swabs taken between the fifth and eighth days of the puerperium, all from women who had had normal deliveries, who had not been examined internally and who presented normal charts. The Rotunda people investigated a similar number under exactly similar clinical conditions. In Edinburgh they found that smears from 60% to 70% of the swabs contained streptococci, in Dublin they found that in 54% streptococci were present. Under conditions pervailing at these famous hospitals it is safe to rule out extrinsic sources of these infections and the first question is where did these microbes come from, from the vagina, the bowel or the blood stream? The second question is what makes for resistance as obviously many patients invaded do not present the picture of infection.

At St. Thomas's Hospital, Lockhart investigated the vaginal flora of one hundred women just prior to labour. He found streptococci present in forty-eight and of these 18% showed signs of pyrexia in their puerperium. Of the fifty-two who did not harbour streptococci only 1·9% developed fever later. Had Lockhart's figures read as fifty infected and fifty non-infected, the percentages would have worked out that he had among the one hundred women ten of pyrexia and nine of them belonged to the first class and therefore their fever could well be accounted for without invoking the aid of any external sources of infection. Lockhart concluded that in his opinion, intrinsic infection was more frequent than extrinsic and added that from questions answered during these investigations he believed coitus late in pregnancy was a frequent source of the microbes lying latent in the vagina.

I shall not deal further with research reports; those of you specially interested have doubtless read these for yourselves. For us general practitioners it is enough to know that until the findings of these three famous schools can be disproved, autogenous infection not only becomes a recognized possibility, but in Lockhart's opinion the predmoinating type of infection.

It may remove the stigma of careless asepsis to transfer the blame from extrinsic to intrinsic microbes, but it does not in any way lessen the responsibility of the obstetrician. Now we have to bear in mind not only external infection, but the microbes which are certainly in the adjacent bowel, are often in the vagina and are probably also in the blood stream. Sterile linen, sterilized gloves and all the disinfectants of the pharmacopœia will fail occasionally, if we do not guard against the elements of shock and exhaustion or see that by the use of hæmostatics after labour we keep the uterus tightly shut and the mouths of the sinuses and lymphatics well closed. In fact the new conception of sepsis demands that we go back into the antenatal period and eliminate all conditions of overwork, anxiety *et cetera,* which may lower the woman's power of resistance as a parturient subject. This is more easily preached than done, for firstly we have no idea just what conditions do lower resistance and, if we did, it would be another question to make the modern woman avoid them. Half of present day civilized mothers are burning life's candle in a wild rush of competition and ambition and the other half are doing the drudgery by day and rocking the ex-baby by night.

The biggest question of present day obstetrics is what constitutes resistance? Sir Ewen Maclean accepting the gift of £10,000 for obstetrical research in Wales has been reported in a recent journal as saying: "There is great need in obstetrical circles for research into conditions which make for and factors which vary resistance to disease."

This is a pithy sentence and I think sums up the perplexities of many general practitioners. Nothing but remarkable powers of resistance can protect some of the back street emergency confinements we have to attend in a welter of filth. Why these same powers of resistance do not stand to our aid in our better class and normal deliveries is the bitter problem of our work.

For instance, we isolated the bacillus of gas gangrene from one uterus in a case of criminal abortion and contrary to all laws and expectations the patient never turned a hair. One of my first cases in private was a case of concealment of birth. I had to conduct the second stage of labour in a fowl house six inches deep in bird droppings, yet this patient was up and about her work in four days. Not long after that I undertook, in the kindness of my heart, to confine for nothing a woman who had a blind husband and ran a small store on commission to keep a roof over the family head. I took her into my private hospital, gave her every aseptic care and after a quick labour and spontaneous delivery, she rewarded me by having a rigor on the tenth day and going on having rigors till she died on the twenty-fourth day. Being young, true to text and ultra-conscientious I immediately emptied my hospital of maternity patients as soon as this patient whom we shall call Mrs. A, had a rigor. I kept away several patients due and generally did the right thing so thoroughly that all the town soon heard I was closed for sepsis and all

the patients due in the next three months made other arrangements!

When the mischief was done, my obstetrical reputation well blasted and the hospital account bankrupt, I got definite advice from another practitioner in the town that he had treated Mr. A. for an acute attack of gonorrhœa inside the last five months. This threw another complexion on Mrs. A.'s fever, but professional etiquette and common decency toward a dying woman made one keep one's own council even at the cost of a few hundred pounds and the ruination of one's obstetrical reputation that cost one's spirit more. I am older and wiser now, but still reflect that one must be either a fool or a philanthrophist if one aspires to build up an obstetrical practice under present conditions in New Zealand.

Later on came another patient with gonorrhœa, Mrs. B.. Frankly she admitted it and was afraid of sepsis in consequence. Realizing by now that one could gamble a little on the inconsistencies of infection I promised to take Mrs. B. into hospital. But this time she had to pay for it and pay well. I had a complete plan of isolation ready for her and took the precaution of notifying the Health Department three months ahead, advising them I did not propose to shut the hospital even if her temperature was raised and kept a duplicate copy of the letter. These precautions taken, the logical sequel of the story is that Mrs. B.'s temperature did not reach 37·2° C. (99° F.)! When we know "the factors which make for resistance and the conditions which vary resistance to disease" I shall have solved the problem of Mrs. A. and Mrs. B. and not till then will you and I approach our patients with any measure of certainty that good work will invariably be followed by good results.

To those of my audience who still have convictions on the error of endogenous infection, I would commend the last message Dr. Fothergill has left us. It is an extract from *The British Medical Journal* of March 6, 1926 and reads:

> In ordinary gynæcological work there are plenty of cases of pelvic peritonitis, tubo-ovarian abscess and acute recurrent attacks of pelvic inflammation in which no one would suggest that the infection had just arrived from the outer world by the vaginal route. Dermoid cysts, ovarians with twisted pedicles and fibroids with impaired blood supply often get infected from the bowel or by the blood stream. What is there peculiar to the puerperal state which can prevent these occurrences? On the other hand the pelvic structures are often bruised during labour and thus all the more likely to be infected from endogenous sources during the puerperium than they are at other times.

Now a word or two about the value of statistics. Both Professor Munro Kerr, in Glasgow, and Professor Werner, of Vienna, said to me: "Statistics between different areas of our home countries are not comparable, much less the statistics of different countries" and every obstetrician we met in England expressed the same opinion.

Concerning morbidity statistics Dr. Hendry, head of a hundred-bed maternity hospital in Glasgow, said: "They are not worth the paper they are written upon, see here and here" (and he took down in all six charts from the wall), "by the British Medical Association standard all these cases should be reported as cases of morbidity, but under the charter of this hospital I am not compelled to notify anything and as I know that the general returns of this area are fallacious, I do not bother to add mine to the farcical compilation."

You have probably heard the definition of statistics, accredited to the late Lord Roseberry: "There are three kinds of liars, there are liars, there are damned liars and there are statistics." Let me show you how true this criticism may become. In Great Britain they may make some effort to compile their morbidity rate, but a few big hospitals out of their control invalidates the whole returns. In New Zealand, on the contrary, we are so tiny that it is easy to trace our patients and we have such a zeal for statistics that we collect them from unwilling ones at the arm of the law. Moreover, every maternity hospital in the land is under Government licence and is compelled to report every case in which the temperature rises to 37·8°C. (100°F.) or more on more than two occasions in the puerperium. Now read the *British Medical Journal* of November 20, 1926, and you will see what happens. Under the title of "The Health of New Zealand" a critic reviews the last annual report of our Director-General of Health. After commenting on the birth rate, the infant mortality rate and so forth, the critic goes on to say: "The number of notifications of septicæmia, however, is very high, 11·93 per 1,000 births being returned as puerperal fever." Now in the year reviewed here, I happened to notify four cases of fever in the puerperium. Not one of these could merit the title of septicæmia, one was a frank case of recurrent cystitis and another was a case of alveolar abscess which was settled by a dentist pulling two offending molars, yet this case gets reviewed in England as a case of septicæmia! By all means let us have statistics to tell us among ourselves what are our losses and what are our septic rates, but when these returns are sent overseas, we might attach an explanatory rider to the effect that our mortality returns are singularly accurate and our morbidity returns are singularly inaccurate, because the obstetricians are not deemed capable of judging whether the fever is of uterine origin or not and have to report every fever in the puerperium whether its focus be in the mouth or on the sole of the foot.

You, Dr. Hooper, made reference in your presidential address to the difference in the mortality returns of Australia and Holland. These returns, if you will excuse my saying it, are not comparable. I was so interested in the matter of statistics that I journeyed to Holland in 1925 where the Health authorities gave me every facility to ascertain how their returns are collected. I hold their own written statement that they doubt if their actual losses are as creditable as international statistics suggest, which statement goes on to say that they know a small percentage of cases are returned, for instance

as death due primarily to pneumonia, whereas the true record should be death due primarily to parturition and secondly to pneumonia. Moreover, they told me that puerperal fever was not a notifiable disease with them, that they knew nothing of a case going wrong till they got the death certificate and that on receipt of that they instituted no inquiries and that none of the maternity hospitals was under any government supervision at all. These interesting sidelights on Dutch methods are sufficiently eloquent to speak for themselves and the only possible conclusion is that, though we all use the same Bertillon system of compiling our returns, once these figures reach our central offices there are such profound differences underlying the methods by which the initial figures are collected that the final returns as standards of comparison are utterly useless.

Statistics can be good servants inasmuch as they warn us how many valuable lives we are losing in our own country, but good servants become bad masters when by fallacious comparisons we raise a mare's nest of public anxiety and cause bitterness and dissension amongst members of the profession.

Finally a few suggestions as to ways and means whereby practitioners may help to remedy the situation pending the time research workers will give us greater light on the problems of resistance and infection. Firstly we can raise our voices in protest at the inferior place given obstetrics in most of our teaching schools and in New Zealand at the inadequate facilities for practical work. My own practical training in normal midwifery was a hasty call out of some surgical class, a scamper down the road just in time to deliver the head under the supervision of a sister. As I learnt later, this was not a training in normal obstetrics at all and with all respect to the lady who taught us, I do not think it was uplifting to the status of obstetrics that this should be the only branch of our medical training in which we were taught by a nurse. In several schools in Britain I found the students in residence following their tutor round all day and generally steeping in an atmosphere of obstetrical lore. It is training like this that we need to save us some of the desperate struggles that characterize our earlier efforts in private practice with abnormalities. Despite what I said previously about the inconsistency of fever generally, I am sure I got a worse percentage in the years during which I was buying in practice the deftness and gentleness I should have learnt as a student. Secondly, we should protest at the environment in which we are often asked to conduct our work. We could, if we liked, put a stop to this scandal. This is an instance in which Australia and New Zealand might well cooperate in a campaign of public education wherein we demand a clean house and a trained nurse or failing these a suitable hospital. As it is, the new graduate's aseptic conscience is horrified at the conditions in which he is expected to conduct a major operation such as labour. Yet, while his colleagues in the town are content with the Gamp, the unshaven vulva and the double bed, he is more or less compelled to fall into line to the detriment of his own self respect and the ruination of his aseptic conscience. Thirdly, we should combine to raise the standard of fees. Present rates are in no small measure responsible for bad midwifery and unnecessary fevers. As they are now, maternity work is the strong man's worst bogey and the small man's best fee and we cannot afford to have our strong men giving up the practice of midwifery.

At the rate of four or five guineas per case no man can afford to specialize in obstetrics; he must do general practice as well. Too often he is away at the opposite end of his beat when wanted urgently for a confinement or else he comes to an abnormal case at eleven o'clock at night, so worn out with the rush of a heavy day's work that it is a physical impossibility for him to give his patient the skill he might otherwise render. I fully agree with Dr. Hooper that the public should and would pay better fees if we proved that a better service is available. Henry Ford has demonstrated that if one gives the service, the public will pay the bill. The majority of patients we attend in private practice is in homes wherein we see gramophones, fur coats and motor cars and as most couples are not going to have more than three or four children in their whole married life, they might well learn the value of children, the perils of childbirth and pay eight to ten guineas for its safe and leisured conduction.

Then we must adopt some line of education so that the public will comprehend our move toward better environment and higher fees. As we have given false impressions concerning the responsibility for the occurrence of fever, we have now no other course than to correct the error. This is necessary, not so much for purposes of self defence, but in order that our patients may have that necessary trust in us without which it is impossible to do good work. We ought to teach them that labour properly conducted involves team work. To my mind every labour conducted in the left lateral position needs one sister handling the anæsthetic and applying pressure on the fundus if requested, one assistant holding up the right leg and the obstetrician absolutely free to mind perineum and anus. All forceps cases require four workers, the extra assistant being employed to raise the right gluteal fold with one hand and to hold in the other hand a small swab ready for any bead of mucus that may appear in the widely opened anal orifice. Such a conception of the bare necessities insists that all *primiparæ* go into hospitals or else we should tell them at the outset that we must engage a colleague to give them the anæsthetic if they insist on delivery in their own homes.

It is time we gave people to understand what serious possibilities surround every case. We get no thanks and reap endless difficulties as a result of belittling the dangers of parturition. At present people think we are nervous or incapable if we ask for assistance and it is high time we taught them

that it is the good man who asks for help before, not after, he gets into difficulties.

In my own technique I set store by the uterine membranes as a help against sepsis. I have had no reason to fear internal examinations or corrective manipulations of position since I adopted the use of tincture of iodine as the last thing I dip my gloved hand into prior to introducing it into the vagina. But, having a wholesome respect for the septic possibilities of the vagina my hand has to pass through, I dread a manual removal of an adherent placenta for then the membranes no longer stand between bacteria that may have eluded the iodine on my glove and the open mouths of the uterine sinuses.

In the present state of knowledge I believe it wise to use pituitrin and intramuscular injections of ergot freely irrespective of whether the patient is bleeding heavily or not. Anything that will keep the uterus firmly shut in the first few hours must be an aid against bacterial multiplication in a blood clot media and extension from there into the open mouths of veins and lymphatics.

I think it is more scientific to convert posterior presentations into anterior presentations after a reasonable trial than to wait till the patient is worn out or till you find eventually that a retraction ring has formed. These are circumstances in which we have to strike a wise balance between meddlesome midwifery and a conservativism that reduces our patient's powers of resistance.

It takes some courage to stand up in New Zealand and say I use forceps often for *primiparæ* in conjunction with scopolamine and morphine for pain relief. But my forceps are invariably the low application used merely to fix the head in the perineal outlet. As I use them they appear to me to be truly prophylactic. My infant record will stand close inspection and my mothers at the termination of the third stage of labour all have slow, full pulses averaging eighty beats per minute. Forceps rightly used can, I think, be a prevention against sepsis, just as much as forceps abused can be a source of sepsis.

In conclusion, let me submit that unassisted labour is a major operation the patient performs on herself, which operation involves a large breach of surface in the deep recesses of a potentially septic canal. How far the changes known to occur in the blood plasma, the protein particles and the parametrium of the pregnant woman are defensive preparations and how often these defensive forces break down under the artificial conditions of modern life are still matters of scientific uncertainty. Only one thing is certain; modern midwifery presents dangers and difficulties which call to the best and bravest of our graduates to make it their life work and until they do this, we shall make little progress.

DR. H. K. CORKHILL (Wellington) said that he was in keen sympathy with many of the remarks which Dr. Gordon had made.

That the whole body of general practitioners—and most of them in New Zealand were in that category—should have fallen under some suspicion in the public mind in respect of the practice of midwifery, as the result of certain public utterances, was bitter to those who were earnestly striving after improvement. But in spite of that he could not but feel convinced that the lines along which they were working in New Zealand were fundamentally the right ones. It was in the method of their application in individual cases that the irritation sometimes lay.

He could not agree with Dr. Gordon in her enthusiasm for the newer idea of autogenous infection as the solution of the still recurring problem of sepsis and the implied suggestion that they had more or less come to the end of progress along the older lines.

That there was hope of help following research work in regard to autogenous infection and resistance, he believed to be truer and probably this would explain a number of their baffling cases.

He still maintained that in their mass attack on the problem from a practical point of view, the important considerations were:—

Improvement in the training of nurses.

Improvement in medical training and as a corollary, improved medical practice.

The raising of the standard of our hospitals and maternity homes.

The furtherance of ante-natal supervision.

They were undoubtedly attaining improvement in the training of their maternity nurses. They had a body of nurses who would compare favourably with those in any part of the world. Would that they could make the work sufficiently attractive to keep more of their generally trained nurses in this branch after they were certificated.

In regard to the training of their medical students he was not in a position to speak, but he gathered from Dr. Gordon's remarks that there were still difficulties to be overcome. It was his impression that the advance in the training of nurses had outstripped that of the medical student.

That there was a necessity for the raising of the standard of our hospitals and maternity homes, was a measure that no thoughtful practitioner amongst them could dispute.

In regard to ante-natal supervision as conducted in New Zealand, he pointed out that this concerned itself in a very practical way with the question of raising resistance, which Dr. Gordon had stressed. Surely the teaching of hygiene, the search for labial infection, for dental sepsis, pyelitis and cystitis, gonorrhœa and the treatment of these conditions and the correction of many conditions which might lead to difficult labour—all these fulfilled the requirements.

It seemed to him a very suggestive thing that the figures for the larger towns in New Zealand were considerably better than those for the rural districts. It could hardly be claimed that the city population had as a whole any personal advantage. In what respects then were the towns at an advantage? It would appear that nursing facilities both as regards personnel and homes were better, that the average of obstetrical skill was probably higher, and that there was more systematic ante-natal supervision. Was it not reasonable to suggest that these were the factors which still called for consideration?

In regard to the question of notification, he did not think that the position was quite so bad as Dr. Gordon had pictured.

It was true that all cases in which the temperature was raised over 100 degrees on more than two occasions, must be notified by the licensee of the hospital, but it was not necessary for the medical practitioner to notify the case as sepsis, unless there was some resonable suspicion. He believed that notification was a valuable measure provided that it was followed up with moderation and common sense. It had been his experience that an honest adherence to the regulations in respect of notification of temperatures met with a reasonable response on the part of the Medical Officer of Health and had not necessarily entailed closure of hospital.

Concerning the suggestions which Dr. Gordon finally made, he was in entire agreement.

Particularly was he convinced of the importance of the management of the third stage—and also of the puerperium—in respect of adequate uterine contraction and drainage.

In conclusion he would repeat his conviction that the fundamental principles on which they were working were sound and that there was still room for improvement along these lines.

He was perfectly certain that more accuracy on the part of themselves as general practitioners in respect of the duties required of them—notification *et cetera*—would soon be met by a more moderate attitude on the part of their officials and with happy results.

SECTION V.—PREVENTIVE MEDICINE.

FATIGUE IN INDUSTRY.

By J. S. PURDY,, D.S.O., V.D., M.D., C.M., D.P.H., F.R.S.E.,
Metropolitan Medical Officer of Health, Sydney

A remarkable feature during the earlier years of the war, when in England it was necessary to concentrate on the organization of munition works, was that for a time by mutual agreement of employers and employees the restrictions as to the hours of work were withdrawn. Men, women, young persons, boys and girls all for a time worked at high pressure with the stimulus of national necessity backed up by high remuneration.

What was the result? It was found that such extensive concentration for long hours on work did not pay. It was found that Sunday labour, that is the lack of one day's rest in seven, did not pay.

Certain employers who kept a tally of output, found that so far from a seven days' working week increasing the output, after the first few months it declined.

There then was resort to six days' work. It was found, however, that the considerable increase of overtime was telling on the workers, as evidenced by the amount of lost time from sickness. With improved management of the factories it was realized that more work was actually done in an eight-hours' day than in a ten-hours' day.

The appointment of a scientific research committee to review the operations of munition workers and the collection of data as to the occurrence of fatigue, showed that the output in certain industries followed the same curve as that of a muscle stimulated by a galvanic current. It was found that just as Nature observes rhythm and prescribes periods of rest, so to attain maximum efficiency in industry it was necessary to observe as closely as possible the prevention of excessive fatigue beyond the point recoverable by a reasonable period of rest.

The old adage of "all work and no play makes Jack a dull boy" was again exemplified in industry. It was found that in mechanical industries the output increased at a certain period of the day and then declined, then after an interval for a meal, again increased, then declined and then in some industries increased for the last hour in an eight-hours' day, but in a ten-hours' day steadily declined.

Another interesting point was that the incidence of accidents where there was machinery, increased as the period of fatigue was reached.

Fatigue has been defined as "the sum of the results of activity which show themselves in a diminished capacity for work."

Activity must precede fatigue and a diminished capacity for work is its measure. A person may suffer from fatigue without being conscious that his activity is decreasing or that there is a failing in the quantity of his output.

Professor Chapman, of Sydney, in a recent address on industrial hygiene stated: "The physiologist regards as fatigue those changes brought about by muscular work which disappear after the cessation of work; as undue fatigue those changes which are more or less enduring as a result of work. The number of days of absence from work may afford evidence of the onset of undue fatigue."

My position as City Health Officer of Sydney has afforded me the unique opportunity of studying the much discussed subject of the relationship of hours of employment as a material determining factor in the production and reduction of fatigue, according

TABLE I.

Period.	Sickness.		Accidents.		Total.	
	Number of Illnesses.	Rate per 1,000 Employees	Number of Accidents.	Rate per 1,000 Employees.	Number of Illnesses and Accidents.	Rate per 1,000 Employees.
November, 1919, to October, 1920	1,532	486.9	448	141.6	1,980	628.5
November, 1920, to October, 1921	1,529	440.3	438	131.5	1,967	571.3
November, 1921, to October, 1922	971	273.1	439	123.7	1,410	397.6
November, 1922, to October, 1923	1,575	441.1	475	133.3	2,050	574.4

TABLE II.

Period.	Sickness.		Accident.		Total.	
	Number of Illnesses.	Rate per 1,000 Employees.	Number of Accidents.	Rate per 1,000 Employees.	Number of Illnesses and Accidents.	Rate per 1,000 Employees.
1322	—	357.6	—	138.6	—	496.2
1923	793	480.5	229	138.8	1,022	619.3
1924	738	444.0	234	140.7	1,182	584.7
1325	640	328.8	230	118.2	870	448.0

to the number of hours worked or the effect of two days' rest per week in contrast to one or one and a half days.

Mr. E. W. Adams, Chief Clerk, has compiled records of the absences from sickness and accidents for all employees for the past seven years. From 1920 to 1925 there were three periods during which the employees of the City Council worked forty-eight hours each week and three periods which they worked forty-four hours each week, there being no Saturday work.

Whilst the graphs show very little difference with regard to accidents, the slight increase in the forty-eight hours' week being due to a proportionate increase of time exposure, there was a definite reduction in the absences from sickness.

The records for the year previous to the operation of the forty-hours' week and for the year subsequent to the reversion to the forty-eight hours' week, are given in Table I.

In the first and second periods there is little difference, but in the second year the sickness rate per thousand employees shows a decrease of 46.3 or 9.4% and that of accident 10.1 or 7.1%, the total decrease being 56.7 or 8.9% for the period without Saturday work.

With regard to the second period, which was the first year of the operation of the reduced hours, during the months of July and August there was an epidemic of influenza affecting 197 and 152 employees, whilst in the corresponding months for 1920 the numbers were 109 for July and 35 for August. The reduction in the total absences being 231 or 38.8%.

In the fourth period, embracing ten months of the reversion to the forty-eight hours' week, there was a great increase on the previous period approximating the totals and rates of sickness and accident for the year preceding the introduction of the forty-four hours' week.

The totals both of sickness and accident are higher than in any of the previous periods, whilst the rates also are higher than for the two periods of reduced hours.

The rate of sickness increased 168 per thousand employees or 71%, that of accidents 9.6 or 7.7%, the increase in the total being 176.8 or 44.4%.

The amount paid by the City Council for sickness and accidents was *per caput* 96s. in 1920, 122s. 7d. in 1921, 83s. 3d. in 1922, 105s. 10d. in 1923, 110s. in 1924.

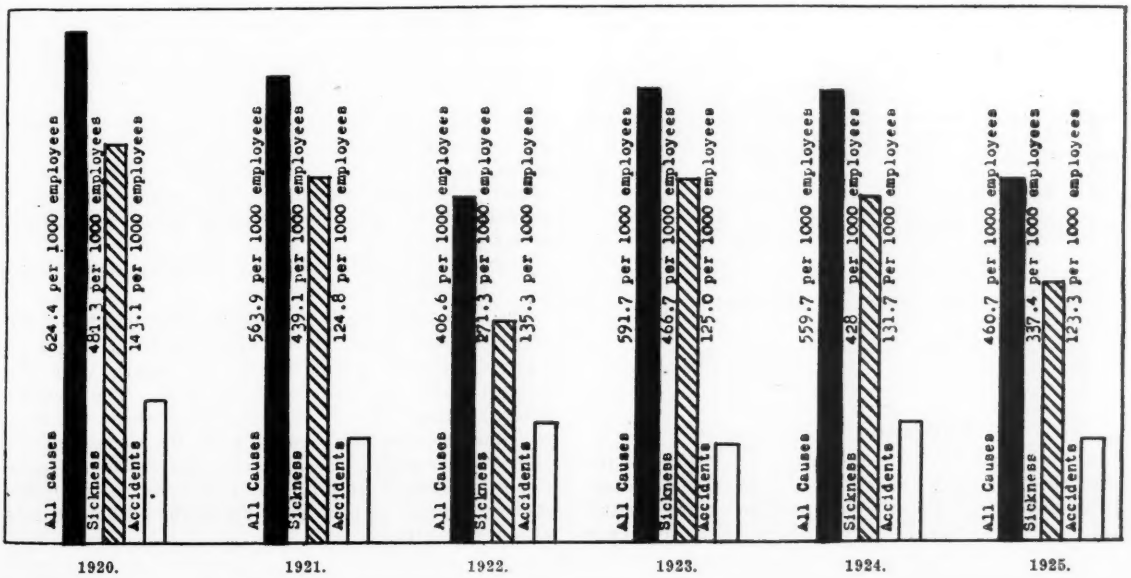
Table II. compiled later shows again the great reduction in relation to the Electricity Department.

It will again be noted that in 1922 and 1925, the years recorded with no Saturday work there is a considerable reduction of sickness as is obvious by a glance at the graph. The forty-four hours' week came into operation on February 10, 1925.

The above data, the earlier figures of which were placed before the Australian Health Congress, Melbourne, and the Royal Commission on Health, are of extraordinary interest in view of the double change in the hours of work during the past six years.

The figures confirm the experience during the war of one of the largest munition factories in England, at which 70,000 workers are employed which in consequence of the number of absences from work gave its employees a whole holiday on Saturday instead of a half day. As a result the absences were diminished by 50% and the firm has continued the innovation.

There was neither a loss in output nor a reduction of efficiency whilst there was less sickness and accident.



The fact that a man knows that he has two days' leisure at the end of the week often makes him remain at his job until Friday and resist an inclination to report "sick"; moreover, for the same reason, men come up on Friday to sign on again to start work on the following Monday, instead of waiting until 9 o'clock on Monday morning for medical examination, which sometimes meant that they do not start until the Tuesday.

A reduction of the amount of overtime worked is probably also a factor in the reduction of absence from sickness and accident.

I had previously noted that when owing to certain circumstances it has been necessary for men to work considerable amounts of overtime and in some cases to do double shifts, there were unusual numbers of cases of neurasthenia and gastritis especially in the summer months.

In some cases men were absent through sickness, largely the result of over-fatigue, treble and quadruple the time they worked overtime.

It was gratifying to note a considerable reduction in neurasthenia as a cause of absence in 1925.

PROFESSOR C. E. HERCUS (Dunedin) was impressed by the convincing graphs in Dr. Purdy's paper which bore out the findings of the Royal Commission. From student work alone he held that there must be proper intervals of rest for recovery in order that the best results might be obtained. He hoped that investigations begun by Dr. Purdy would be extended in view of the large population in Australia.

DR. T. MCKIBBIN (Wellington) stated that primitive man had not worked at the pressure of the present generation. Longer rest periods were needed in modern times. He agreed that there was a definite rhythm in industry and that Saturday half-day was ineffective as a working day. The forty-four hour week would give a better rest period.

DR. J. S. PURDY in reply pointed out that the reduction in the amount of absenteeism of Sydney City Council employees was perhaps partly due to the fact that a man would hang on until Friday instead of "reporting sick" so that he could have forty-eight hours of complete rest. The consensus of opinion was that an eight-hour day gave a better output than ten hours, but whether six hours would be better than eight hours in the end result was very debatable. Mrs. Osborne, of Melbourne, had proved to the satisfaction of the Arbitration Court that it was not beneficial either to industry or to employees to employ women in factories more than forty-four hours weekly. For all classes more recreation facilities should be provided in the large cities, more open air spaces, that longer rest periods might be beneficially used.

SECTION VII.—OTOLOGY, RHINOLOGY AND LARYNGOLOGY.

PARANASAL SINUSITIS IN CHILDREN.

By ERIC GUTTERIDGE, M.D.,
Honorary Aural Surgeon, The Children's Hospital, Melbourne.

The Development of the Sinuses.

In the third week of embryonic life appears the primordial cranium as a thickening of the mesenchyme investing the anterior portion of the medullary canal. The basal part of this cranium developes into chondro-cranium. The lateral basal plates develope as the peri-otic capsule, ventrally form the mesethmoid plate (vomer *et cetera*) and antero-laterally they enter into the formation of the nasal capsule.

The lateral wall of the nose is formed from the fusion of the lateral nasal processes and the maxillary process, the medial wall from the median nasal and globular processes.

From the fortieth day appears a slight folding on the lateral wall representing the inferior concha and a week later superiorly and slightly dorsally the ethmoidal fold and concha.

At the sixtieth day appears a ridge extending postero-inferiorly along the superior curved border of the inferior concha. This is the *processus uncinatus* and shortly afterwards inferior to the attachment of the middle concha appears a projection—the *bulla ethmoidalis*.

The Maxillary Sinus.

Primarily a pouching of the mucous membrane of the lateral wall of the *infundibulum ethmoidale* is evident about the 70th day of fœtal life (Schaeffer).

The pouch may be duplicated, thus accounting for the rare cases of double adult maxillary sinuses or these pouches may fuse with duplicate ostia or a large slit-like ostium.

The early sinus is for a time a narrow cavity in the membranous lateral wall of the nose. It extends inferiorly into the recess formed by the union of the lateral plate of the encircling cartilaginous nasal capsule with the plate of the inferior nasal concha.

The portion of this capsule plate intervening between the maxilla and the developing maxillary sinus sac is resorbed and a direct relationship with the maxilla is established. Further enlargement of the sac and absorption of the body of the maxilla ensues.

In the embryo the *processus alveolaris* of the maxilla which contains the unerupted teeth is in proximity to the orbit and therefore the sinus must be correspondingly small.

In childhood growth of the sinus and resorption of the maxilla continue. By the twelfth month the sinus has developed sufficiently in width to reach slightly beneath the orbit and soon afterwards extends above the rudimentary first permanent molar tooth.

During the second year the sinus passes beneath the infra-orbital nerve.

The dimensions are as follows: The length of the ventro-dorsal diameter at birth is from seven to eight millimetres and up to the eighth year the annual gain is three millimetres. The medio-lateral diameter at birth is from three to four millimetres and the annual gain up to the eighth year in two millimetres. The height or the cephalo-caudal diameter at birth is from four to six millimetres and the annual gain up to the eighth year is two millimetres.

ILLUSTRATIONS TO DR. J. F. MACKEDDIE'S ARTICLE

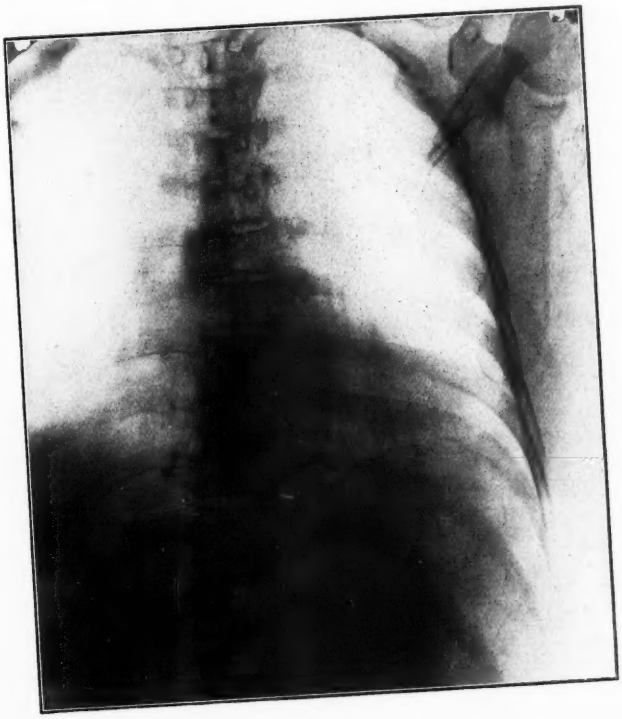


FIGURE I.

FIGURE II.

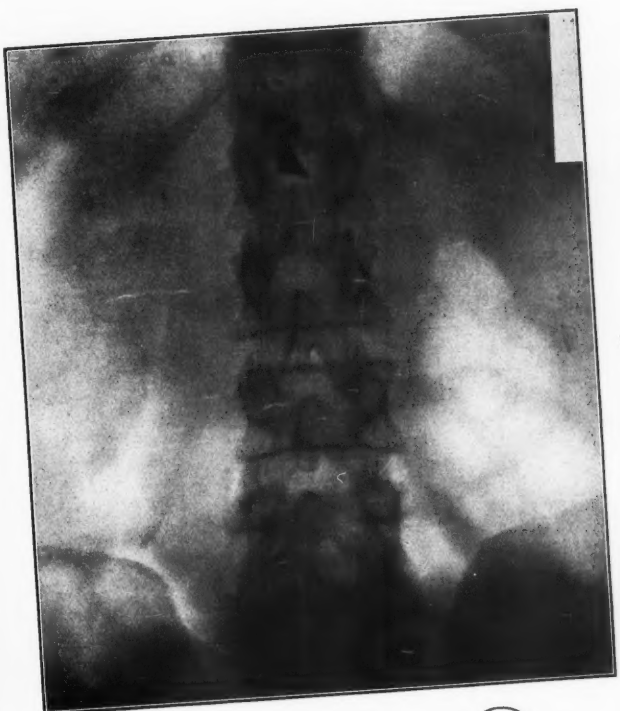
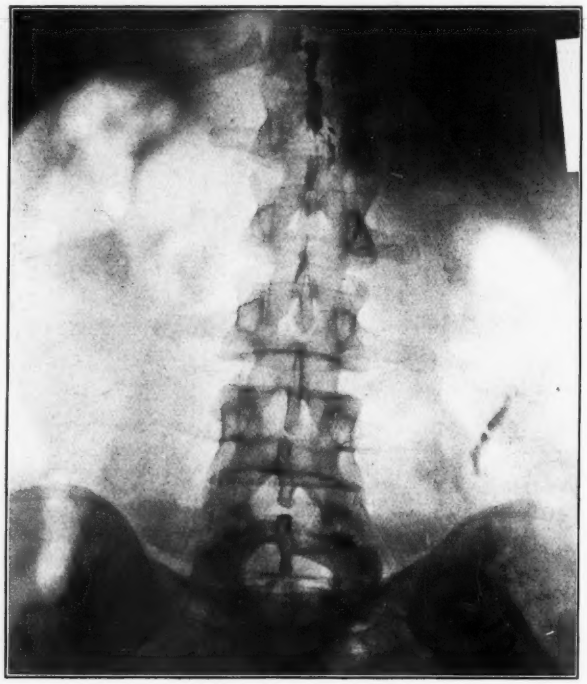


FIGURE III.

UNIV. OF MICH.

ILLUSTRATIONS TO DR. J. F. MACKEDDIE'S ARTICLE.



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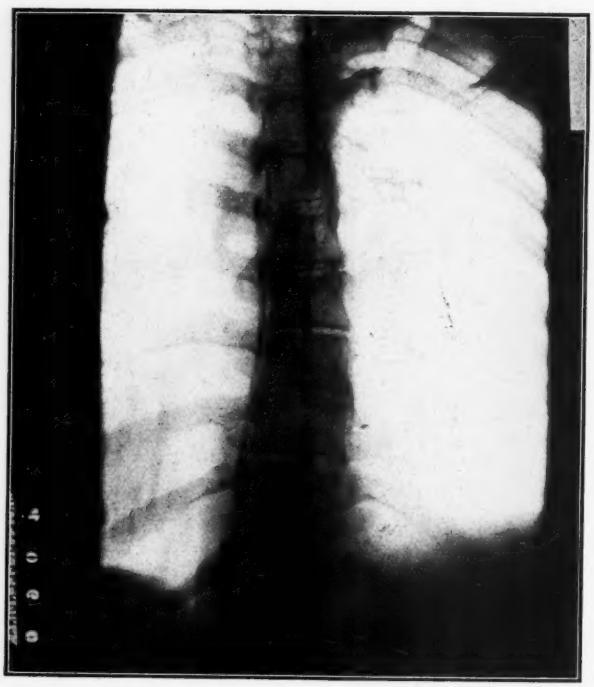

FIGURE V.

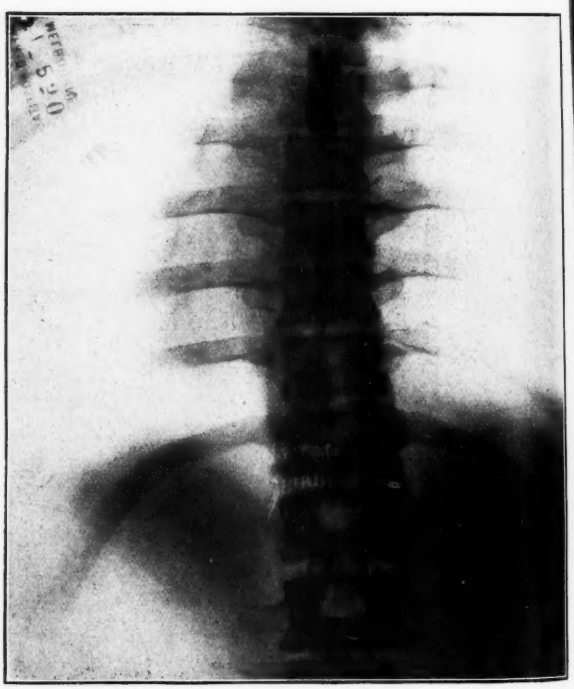

FIGURE VI.

ILLUSTRATIONS TO DR. J. F. MACKEDDIE'S ARTICLE

FIGURE VII.

FIGURE VIII.

FIGURE IX.
The "Lipiodol" Showing up Well in the Cervical Region, but Very Obscure.

FIGURE X.
This shows the upper limit of the "Lipiodol" ascendant before the insertion and descent of the "Lipiodol" descendant. (The blob marked (D) was some heavy "Lipiodol" put in at the same lumbar puncture as the light "Lipiodol" to contrast its action with the light "Lipiodol.")

ILLUSTRATIONS TO DR. J. F. MACKEDDIE'S ARTICLE

FIGURE XI.
This shows the upper limit of the ascending "Lipiodol" (A) and the lower limit of the descending (D).

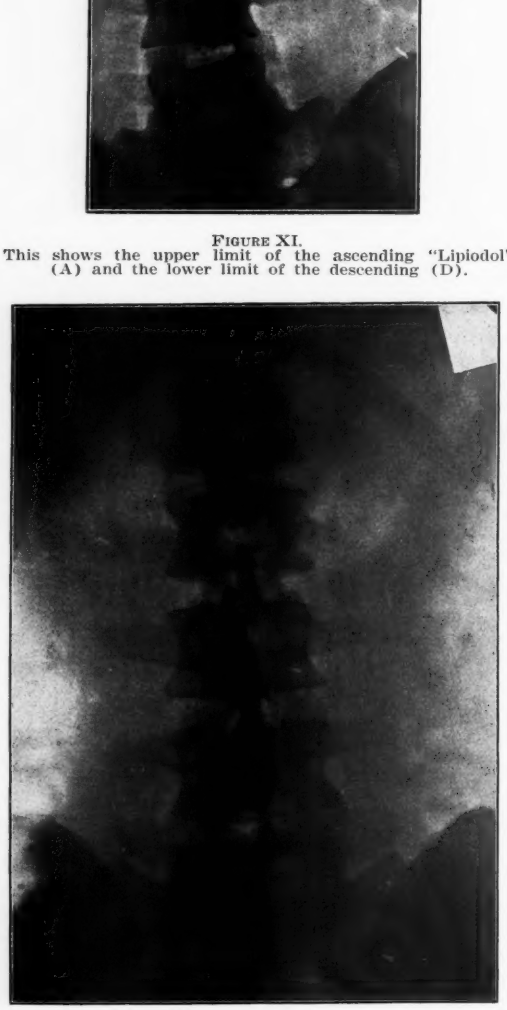

FIGURE XII.
The "Lipiodol" is blocked at the start of its journey.

FIGURE XIII.
This illustrates the "Lipiodol" in the tissue of the neck and not in the *cisterna*.

FIGURE XIV.
This illustrates ascending "Lipiodol" settling finally at the base of the brain in the *cisterna basilaris* and in other subarachnoid spaces.

After the eighth year the sinus advances more slowly in all directions approximating to the adult about the fifteenth year.

Subsequent development is chiefly in the postero-inferior angle which descends as the third molar tooth erupts.

The floor of the sinus in the infant at birth is approximately four millimetres above the nasal floor and does not reach that level until the sixth or seventh year. This has an important bearing in intra-nasal procedures.

The Frontal Sinus.

The naso-frontal region is genetically an outgrowth from the ventral and cephalic end of the middle nasal meatus. This *recessus frontalis* begins to appear in the fourth month, its smooth surface soon shows folds and furrows. These frontal furrows or pits invaginate early and form certain of the anterior group of ethmoidal cells, the so-called frontal cells.

The frontal sinus developes variably: (i.) By a direct extension of the whole *recessus frontalis,* (ii.) from one or more of the anterior group of anterior ethmoid cells originating in the frontal furrows, (iii.) occasionally from the ventral extremity of the *infundibulum ethmoidale.*

The sinus frontalis may or may not be present at birth. The various potential rudiments of the sinus are far advanced, none, however, topographically frontal. These pouches continue to extend their boundaries and the predestined sac ultimately comes into contact with the horizontal portion (*pars orbitalis*) of the frontal bone, resorbs this cancellous bone and grows between the squama of the frontal.

There is considerable variation in growth. However, by the twentieth month the frontal sinus has eroded into and begun to ascend the vertical portion of the frontal bone and by the middle of the third year the *cupola* is above the level of the *nasion.*

When the sinus first invades the vertical portion of the frontal bone, it is nearer the inner than the outer plate, with the result that there is a thin dorsal wall almost wholly composed of compact bone, as opposed to a fairly thick anterior bone of both compact and cancellous (diploic) bone.

Very frequently there is a marked asymmetry of the sinuses. A persistent metopic suture usually precludes the likelihood of development of the sinuses beyond the median plane.

The dimensions are as shown in the table.

Age.	Distance of of Cupola from Nasion in Millimeters.	Height in Millimetres.	Width in Millimetres.	Ventro-dorsal in Millimetres.
6-12 months	2 below ..	2	1·0	3·5
1-2 years ..	1·5 above..	5	2·5	4·5
3-4 years ..	2 above ..	6	3·5	5·0
	3·5 above..	8	3·5	6·0
5-6 years ..				

The Ethmoidal Cells.

The ethmoidal cells are primarily extensions or evaginations of the nasal mucosa from the middle, superior and first supreme nasal meati, that is from the frontal meati directly or the accessory furrows and recesses which configure their lateral walls.

The initial ethmoidal outpouchings are evident in the fourth month of intrauterine life. By the seventh month they form hollow, tubular-like, blindly ending sacs with ostia in communication with the points of initial outgrowth.

In childhood growth continues by a honeycombing of the lateral mass of the ethmoid bone with a definite growth tendency toward the cribriform plate, but, as a result of crowding of the cells this growth is in all directions.

There is early a division into non-intercommunicating anterior and posterior groups originating and draining into the middle and superior meati respectively.

The frontal furrows in the lateral walls vary in number. There may be a total absence and a simple blind outgrowth from the middle meatus. These furrows develope variously into anterior ethmoid cells and in addition in the majority of cases the frontal sinus is formed as an outgrowth from this region.

The *agger nasi* is very commonly pneumatized by the most ventral of the ethmoidal cells developing from these frontal furrows. The *infundibulum ethmoidale* may terminate ventrally and superiorly by dilating into a single ethmoidal cell lateral to the frontal recess and its derivatives or may bud off two or more cells which may become frontal sinuses or form bullar-like swellings on the frontal floor or grow into the *agger nasi* or *processus uncinatus.*

The bullar or middle ethmoidal cells vary from one to five, arising from the supra-bullar or infra-bullar folds, hollow out the bulla and frequently extend to the supra-orbital plate of the frontal or orbital plate of the maxillary bones.

The Post-Ethmoidal Cells.

During childhood the post-ethmoidal cells gradually pneumatize the superior and supreme

Age.	Anterior Ethmoidal.		
	Height in Millimetres.	Width in Millimetres.	Length in Millimetres.
At birth ..	5·0	2·0	2·0
2 years ..	3—9	2—6	2—7
5 years ..	7—8	5—7	5—6
8 years ..	8—11	7—10	6—7

Age.	Posterior Ethmoidal.		
	Height in Millimetres.	Width in Millimetres.	Length in Millimetres.
At birth ..	5·0	4·0	2·0
2 years ..	5—8	3—4	4—6
5 years ..	7—8	7—10	6—7
8 years ..	8—11	7—10	9—15

conchæ and may extend into the supra-orbital plate of the frontal and the infra-orbital of the maxilla. The dimensions are given in the table.

The Sphenoid Cells.

The sphenoidal cells primarily arise in relation with the posterior *cupola* or dome of the cartilaginous nasal capsule from the terminal nasal sinus and in the fœtus is a purely nasal pouch.

By the end of the fourth year the intervening nasal capsule has been resorbed and the body of the sphenoid bone now bounds the terminal nasal sinus (the primitive sphenoidal sinus). Later there is absorption and pneumatization of the sphenoid bone.

The sinus has a tendency to develope more dorso-laterally than ventro-laterally with early thinning of the lateral walls of the sinus and relatively thick septal and dorsal walls. The dorso-lateral walls may come early into intimate contact with the ophthalmic and maxillary nerves and be a potent factor in childhood neuralgias of the trigeminal nerve as early as the third year. The dimensions are shown in the table.

Age.	Height in Millimetres.	Width in Millimetres.	Length in Millimetres.
First year ..	2·5	2·5	1·6
Second year ..	4·0	3·5	2·0
Third year ..	5·5	4·0	2·5
Fifth year ..	7·0	6·5	4·5

Acute Sinusitis.

Paranasal sinusitis in children may be classified as in adults into acute, subacute and chronic sinusitis.

Acute sinusitis which includes the common cold, is frequent in childhood. The narrowness of the nasal passages and the inability to evacuate the discharges, as by blowing and sucking the discharges into the naso-pharynx, accentuate the discomfort, prolong the disease and liability of spread to the remainder of the respiratory tract. These factors become progressively less as the child grows. There is a tendency toward stagnation of the discharges in the sinuses and the development of subacute and chronic sinusitis.

The acute infections as scarlet fever, diphtheria, influenza and pneumonia are frequently followed by acute sinusitis with a special liability to osteomyelitis with external abscess in the orbit (Skillern, Bryan). Skillern states that in infants under two years of age the infection always attacks the tissues as a whole, not only the mucosa, but the underlying soft spongy bone, so that almost the whole side of the nose and often the face is affected.

The rôle of excessive lymphoid tissue in the naso-pharynx and hypertrophied and infected tonsils is a predetermining and accentuating factor. This is amply proved by the beneficial effects of removal of these tissues.

Nasal septa which are sufficiently deflected to cause obstruction of the airway, and enlarged cellular middle conchæ impede the drainage of the sinuses.

Chronic Sinusitis.

Chronic sinusitis and its predecessor, subacute sinusitis, are exceedingly frequent in childhood. For many years it has been recognized that chronic parenchymatous tonsillitis associated with hypertrophy of the naso-pharyngeal tonsil caused a chain of symptoms of nasal catarrhs and obstruction, ear catarrhs and suppuration, reflex and general symptoms. These effects are partly due to the accompanying sinusitis, which may persist after removal of both sets of these tonsils.

The majority of patients whose histories are extracted below had undergone this operation.

Symptoms.

The symptoms include acute coryza, frequent and prolonged, especially in the colder months and never quite clearing, nasal breathing is difficult and noisy, especially at night. The mouth is of necessity partly opened. The rodent face tendency appears. Aprosexia is threatened. These conditions occur even in the absence of adeno-tonsillar enlargement or deflected obstructing septa.

Nasal discharge is complained of in 90%, is in profuse or small quantities, mucoid or purulent or muco-purulent. It may be blood-stained or epistaxis may occur. The discharge may be frôm the posterior nares and may be expectorated or, in younger children, swallowed. The child may snuffle and sniff.

Headaches are chiefly frontal and recurrent and may continue for one to three days with gastro-intestinal disturbances, vomiting, malaise, and so forth. Coryza is an occasional accompaniment, at the time or as a terminal discharge. These headaches are chiefly antral and are then relieved by lavage-puncture.

Eczematous conditions of the nasal vestibule occur from the irritation of the discharge and wiping and rubbing of the nose.

Further down the respiratory tract there is the chronic cough, of harsh, dry and paroxysmal character or with little or much sputum, mucoid or purulent. This cough is very refractory to medical measures. It may be croupy (seven cases) or be a laryngitis.

Acute bronchitis, once or more, is a frequent history (nine cases).

Pneumonia occurred in three patients.

Bronchiectasis with quantities of purulent sputum and unaffected by vaccines, drugs or posture occurred in one patient and was completely relieved by a double radical Caldwell-Luc operation.

Asthma was a frequent relation. It occurred in twelve patients and improved by treatment.

Peri-bronchial fibrosis with hilum shadows and fibro-calcareous glands was reported in 16%. One of these was much improved under lavage.

The tongue is constantly coated, the breath is foul and frequent attacks of tonsillitis occur. There

is vomiting, cyclic, with digestive disturbances, anorexia, bilious attacks with or without headaches. Constipation is common.

Of ocular manifestations iritis and conjunctivitis are frequent, retro-bulbar neuritis rare. Errors of refraction may be much improved by treatment of the sinuses.

Nephritis and pyelitis of childhood include sinusitis in their ætiology (Dean).

Lethargy and chorea and choreiform twitchings of arms and shoulders and head are also seen.

Of aural complications catarrhal or suppurative otitis is frequent and occurred seventeen times in this series, it being bilateral three times.

Signs.

The middle concha is occasionally apparently normal but usually (60%) is slightly pinker and a little blurred in outline. In the later stages of undoubted chronicity there is a darkening of colour to a maroon shade, a greater thickening of the mucosa, which may be harsh and scaly or œdematous and polypoidal. In one case only was there a definite atrophic rhinitis. These changes were not noted to be more marked in the hiatal portion.

Excessive nasal secretion was noted on the floor of the nose, in the *hiatus semilunaris* and medial to the concha. It occurred as strings or mucus-clothes lines, between conchæ and septum very frequently. This secretion was mucoid in 65%, pus or muco-pus was seen in 30%. In some cases pus was visible on the post-pharyngeal wall and was often accompanied by chronic pharyngitis.

The septum was much deflected in 12% and was associated occasionally with cellular enlargement of the middle concha, causing obstruction.

The external nares showed some irritation with thickening and excoriation.

There was frequently an unpleasant odour and coated tongue.

Tonsillar tissue was present in the small proportion of 15%, as most of these cases were referred from surgical clinics where they had been regarded as showing the tonsillo-adenoid syndrome and had been operated. The tonsils were in the majority diseased, follicular and with inflamed anterior pillars.

Similarly hypertrophied adenoid tissue was seen in 12%.

The anterior group of cervical glands were enlarged in 10%, the rosary type.

Aural manifestations were common as active or subsided suppurative *otitis media*, in 20% one ear, in 7% both. There was retraction of the *membrani tympani* in 5%. Deafness of the catarrhal type appeared occasionally.

The mouth was of the "adenoid facies" type in a number of the children with high Gothic palates and rodent incisors. How much of this was due to adenoids was uncertain.

The complexion may be sallow or show a bluish-purple tint or a dark patch under each eye. The hands may be blue and cold, the expression dull or vacant; the child shows little or no energy (Cleminson).

Diagnosis.

The diagnosis rests on the symptoms and signs, on the results of radiography and exploratory puncture. Transillumination in children is not a sure guide. In normal cases in the youngest children the inner horn only of the infra-ocular crescent lights up, though as the years advance the zone of illumination extends until in older children the crescent is complete (Cleminson).

The radiogram has not in children the value that is placed upon it in adults. The developing teeth and the milder inflammation of the mucosa not being so opaque to the rays, detract from its usefulness. It gives valuable information on the condition and development of the ethmoids and frontal sinuses

The final stage in the diagnosis is exploratory puncture. This is done under ethyl chloride The infra-turbinal area is cleansed with alcohol. The antrum trocar passes through the naso-antral wall and connects with a "Record" syringe and sterile water is sucked in and out. Or the patient is allowed to regain consciousness and the antra are washed out by means of a Higginson's syringe in the ordinary manner. A negative result is regarded as of no value. Positive results were noted in 25%, mucus in 6%, pus in 12%. A few threads or shreds of muco-pus in a fairly clear fluid or a large clot of yellow pus may occlude the ostium and require some pressure to dislodge.

The sinuses affected are in descending order of frequency, ethmoidal, maxillary, sphenoidal and frontal (Davis). The diagnosis is based largely upon the radiograms, transillumination and the results of lavage. Probably there is an infection of both antra and ethmoids in the majority of cases. Clinically, it was found that lavage of the antra reduced the congestion in the nose and tended to improve the ethmoidal disease.

Bacteriology.

Bacteriological examination was carried out in a proportion of cases. The pneumococcus and *Micrococcus catarrhalis* were the more frequent, but not sufficient data were obtained to be of value.

Treatment.

1. Dietetic measures include the provision of fat-rich diet, fat-soluble vitamin A in cod-liver oil (Lore). The correction of errors in diet, excess of carbohydrates, deficiency in proteins of the animal group is important.

2. Climatic. Air heavily laden with moisture as in the seaside suburbs has a deleterious effect upon these children and the removal to a higher area where the air is drier and more bracing improves them. On the other hand sea bathing and especially insolation in running about the beaches is of advantage. Similarly badly drained, damp houses on clay soil and in low-lying suburbs lead to frequent reinfections. Overcrowding in the cities

with exposure to dust and contagion have a bad effect. In many of these children a holiday or removal to the country completely clears the nasal condition.

3. Nasal hygienic measures should be used. The children should be taught to blow the nose; the instillation of antiseptics, "Argyrol" (5% to 10%), "Menthol," "Eucalyptol" and camphor in a paraffin oil base or menthol and compound tincture of benzoin inhalations are of value. Haskins gives 0.3% phenol in petrolatum. Nasal douches were not found of much avail.

4. Antral lavage as a therapeutic measure repeated up to four treatments cleared the majority, at least temporarily. Usually both antra were washed out.

5. Where there was marked obstruction of the airway by a deflected septum, this was corrected.

6. Further procedures are in the nature of antral drainage, firstly by naso-antral opening beneath the inferior concha, with as large an opening as possible. This was specially indicated in those cases with much pus on puncture. The Caldwell-Luc technique with retention of the mucosa was the next step. If the mucosa was much hypertrophied or polypoid, it was removed.

7. Uncapping the ethmoids and sphenoids is advocated by Lore where these measures fail.

8. Vaccine therapy was found to be of no effect in the unoperated cases, but as an adjunct to drainage it is of assistance. "Collosal manganese" used in the same manner is recommended by some.

9. The Jansen method of opening the ethmoids is reserved for the intractible sinusites.

The following conclusions may legitimately be drawn.

Sinusitis in children is very frequent and is not sufficiently emphasized in works on pædiatrics or in our specialty. It has protean manifestations and especially attacks the respiratory tract. It may be said that a vast proportion of these diseases owe their origin and persistence to the sinusitis and will be remedied by treatment applied to the nose and throat.

The effects of enlarged and infected tonsils and adenoids are largely due, it is my belief, to the accompanying sinusitis. Removal of these lymphatic infections will cure the majority of these patients. It is generally held that the figure is as high as 80%. In this series 85% had been dealt with in this way and I am practically concerned only with the 20% in whom tonsillectomy had failed to cure the sinusitis.

The reports of the children after a lapse of some months show that 21% were much improved, 30% were improved and 17% were in the same state; 32% were not seen again. This means that of the children concerning whom progress reports have been received 80% were improved or relieved.

Whether these children are permanently cured or their symptoms temporarily abated to return after another coryza or in the next winter, it is impossible to be sure, but the gain to their health and the relief to their lungs and developing nasopharynx is of inestimable value. A similar treatment will probably again be successful.

There still remains a residuum of cases which will require further operative treatment. The Caldwell-Luc operation was performed on 14% of the children and the mucosa was found to be polypoid in 7%.

No complications as meningitis, frontal osteomyelitis or orbital abscess, as noted by Skillern and others, were seen in this clinic.

DR. H. F. SHORNEY (Adelaide) cited the case of a little boy, aged seven years, with severe bronchitis. After an X ray photograph had been taken and a polypoid growth had been found in the antrum, he had performed the Caldwell-Luc operation with cure of the bronchitis. He advocated the Caldwell-Luc operation in antrum trouble, because it enabled the surgeon to inspect the condition of the mucosa. Lavage under a general anæsthetic was said to be practised in America. He wished to know what precautions were taken.

DR. T. A. MACGIBBON (Christchurch) in discussing the anatomy of the sinuses said that there was a diversity of opinion as to the time when the floor of the auxiliary antrum began to be displaced downwards. He doubted the advisability of antral lavage in patients under the age of six years. He agreed that the Caldwell-Luc operation enabled the surgeon to see the mucosa. Polypi and septæ were often seen. There was a prior stage of ozæna observed in children. This was associated with a bluish discharge of a mucoid character; there was no odour at this stage. The condition, however, was the beginning of atrophic rhinitis. A characteristic red nose was frequently encountered in children with chronic sinusitis. He advised washing out the sinuses many times before the surgeon proceeded to operative measures.

DR. W. N. ROBERTSON (Brisbane) said that Dr. Gutteridge had called attention in his important paper to the fact that many bronchial conditions in children were due to sinusitis or bronchiectasis. In his own practice he removed the tonsils and adenoid vegetations and if the condition had not responded to the treatment, he then washed out the antra frequently. He cited a case of a child with ethmoidal disease at the very early age of three years. The bridge of the nose was bulging. He had little faith in X ray photographs for children. They had not given him any real help.

DR. R. PULLEINE (Adelaide) was glad to note that Dr. Gutteridge had called attention to the fact that in some children sinusitis was due to deficiency in diet and climatic conditions. He doubted whether true atrophic rhinitis could be cured by antral lavage or operation. In his opinion atrophic rhinitis was quite uninfluenced by operative treatment.

DR. GUTTERIDGE in reply said that a radical Caldwell-Luc operation was performed if the mucosa was found to be thickened or polypoid. He employed ethyl chloride anæsthesia for antral lavage in children. The trocar was inserted and the child was then allowed to regain consciousness. The head was placed well forward and the antrum was syringed gently. No pain was felt. He advocated lavage four or five times. Then an intranasal opening was made. The Caldwell-Luc operation was the last resort. He held that there was very little ozæna in Melbourne.

ALLERGY OF THE RESPIRATORY TRACT.

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OUR modern conception of allergic disease draws its beginnings from the work of Richet some twenty years ago. In the interval since that time we have had ample demonstration of the value which a

seemingly isolated laboratory observation can have in the understanding and treatment of disease. It is this practical application which I wish to stress in this essay on allergic disease.

Allergy has been defined by Pirquet[1] as a constitutional hypersensitiveness to foreign substances, usually of protein nature. We now know that this hypersensitiveness may result in the familiar clinical symptoms of asthma, hay-fever, urticaria, eczema and certain gastro-intestinal disturbances. In order that I may not wander too far afield, I will limit myself to a discussion of the manifestations of allergic disease upon the respiratory organs only, that is asthma and hay-fever.

Without entering into too great historical detail let me rapidly sketch the high lights of the unfolding of our present understanding of allergic disease from the first isolated observation of Richet.

In 1898 Richet[2] coined a term, anaphylaxis, to designate the phenomenon which occurred upon the injection of a sublethal dose of congestin following upon a prior sublethal administration of the same substance. Theobald Smith[3] in 1903 while working with diphtheria antitoxic serum, observed the same phenomenon in guinea pigs, finding that a second injection of the otherwise innocuous antitoxic serum proved lethal provided a suitable interval had elapsed after the first injection. Rosenau and Anderson[4] in 1906 while working in the Hygienic Laboratory established two important facts concerning the phenomenon of anaphylaxis: first, that the toxicity of a second injection after a sufficient interval is a common property of all proteins; second, that the reaction is specific in nature and in order to obtain such a reaction, the second dose must be of a protein identical with the first. It was Wolff-Eisner[5] in 1906 and Meltzer[6] in 1910 who first suggested that the diseases known as urticaria, hay-fever and asthma resembled the phenomena of experimental anaphylaxis. Soon thereafter the practical application of these observations and theories led to our modern methods of classification, diagnosis and treatment of these conditions.

CLASSIFICATION.

The classification of asthma and hay-fever is for practical purposes an essentially ætiological one as follows:

Demonstrably sensitive: Seasonal (spring or fall), pollens, food; perennial, pollens, foods, miscellaneous, dusts, bacterial (?).

Not demonstrably sensitive: Seasonal and perennial, bacterial, physical agents (heat-cold), psychic.

Pollen allergy usually manifests itself as hay-fever, although asthma is not at all uncommon, the dividing line between the two conditions being far from distinct and both often occur in a single individual either consecutively or simultaneously. For clinical purposes pollen allergy is most usefully divided into three types according to the seasonal character, namely, spring, fall and perennial. The perennial type is found only in tropical and subtropical climates in which pollination continues throughout the year.

Food allergy is the most common cause of perennial asthma and hay-fever. However, a food of seasonal character may produce seasonal symptoms. The most common offenders among the foods are usually those most common in the diet, namely milk, eggs and wheat. Sensitization to foods is most common in children and here we find many sufferers of what might be termed chronic hay-fever in addition to asthma (Piness and Miller[7]). By chronic hay-fever I mean a perennial nasal obstruction, constant coryza, frequent attacks of sneezing on arising in the morning and usually with a history of numerous operations on the nose and throat, for example repeated adenoidectomies. Here the fact had been lost sight of that the adenoid hypertrophy is the result of the allergic disease rather than the cause of symptoms.

Sensitization to animal dander which cling to the hair or feathers shed by various animals is a frequent cause of both asthma and hay-fever (Goodale[8] and Walker[9]). The most common dander that produce allergic symptoms, are as one would expect those of the common domestic animals such as the horse, dog, cat, rabbit, goose, chicken, cattle, goats and sheep, although theoretically there is no reason to believe that contact with any unusual species of animal might not result in a manifestation of sensitization to its dander.

Under the heading of miscellaneous agents producing asthma and hay-fever in allergic individuals, the most important is orris root, the base of most perfumed cosmetics. Another important agent is pyrethrum, the base of most insect powders.

Bacteria as ætiologic factors in producing the symptoms of allergic disease have long been under suspicion (Walker[10]). However, no one as yet has demonstrated beyond a doubt that they may be considered as allergic agents of the type of pollens, foods and animal dander. Bacterial invasion undoubtedly produces asthma, but as a result of inflammation rather than sensitization.

The idea of specific sensitiveness to house-dust as a cause of asthma and hay-fever was advanced by Cooke[11] in 1922. This investigator recognized the possibility of the dust containing proteins from various and sundry sources such as from a dog or cat in the house, but claimed that such is not always the case, that there is some substance specific to house-dust and of unknown origin. My experience has been that the specific substance in house-dust can frequently be traced to some definite source thus raising the presumption that in those instances in which this has not been possible it has been due to an insufficiently extensive search.

Duke[12] has described and we are all familiar with the production of the symptoms of asthma and hay-fever by such physical agents as heat and cold. Duke is pleased to term this a manifestation of physical allergy. It is my opinion that a physical agent may be a precipitating factor yet scarcely an ætiologic agent in the production of symptoms. In many such cases it is possible to demonstrate a sensitization to foreign protein. In those in which

this is not possible, we should hardly say that there is a sensitization to heat or cold, reserving this term sensitization for use in its usual immunological sense.

DIAGNOSIS.

The diagnosis of allergic disease is based on the facts elicited from the patient such as the family history and clinical history, physical examination, X ray examination, laboratory studies and protein skin tests. All these must be carefully reviewed and in themselves should prove sufficient to make possible a diagnosis of allergic disease.

These diagnostic points will be taken up in order and discussed from the point of view of the student of allergy.

Family History.

A history of allergy in the family of the patient is obtainable in about 50% of cases (Adkinson[13] and Spain[14]). The importance of eliciting a careful family history is therefore obvious.

Personal History.

Age of onset is an important factor to the extent that as age increases, the frequency of development of allergic disease decreases. It is rare to observe the onset of allergic disease past the age of fifty years. However, in many patients of advanced age or of the age beyond which one would expect a development of allergic diseases, such as hay-fever and asthma, a history of eczema or other manifestation of allergy in infancy or early life is often obtained. Other important points to be brought out in the personal history are with regard to the seasonal character of the illness and effect of change of residence. Of the frequency of so-called "head colds" and of periodic attacks of bronchitis of non-asthmatic character with or without fever, of repeated operations on the nose and throat (Piness and Miller[15]), of the character of bedding, furniture, rugs and cosmetics used, of the effects noted from the ingestion of certain foods, occupational pursuits, contact with certain animals or certain objects and of the effects noted in attempting to obtain relief from such drugs as adrenalin, ephedrin and belladonna. All these are important and any one of them may furnish an invaluable lead that may mean correct diagnosis and proper treatment to the benefit of both the patient and practitioner.

Physical Examination.

A careful general physical examination is as much of a necessity in the proper care of the allergic patient as in other medical conditions. The physical findings in asthma are too familiar to warrant discussion further than to emphasize the necessity of differentiating them from those of pulmonary tuberculosis, myocardial insufficiency and foreign body in a bronchus which I have not infrequently seen with the symptoms of asthma. On the other hand completely negative findings are not at all uncommon during intervals free from symptoms. It is under the latter circumstances and particularly when we may speak of the symptoms as being doubtful in character that a careful examination of the nose and throat by a competent specialist is an absolute prerequisite. Characteristically (Montgomery[16]) he will find here a greater or lesser degree of engorgement and lymphadenoid hypertrophy with its usual sequelæ, namely polyp formation and enlargement of turbinates, tonsils and adenoids, explaining why many allergic individuals exhibit the signs and symptoms characteristic of sinusitis, otitis media and nasal obstruction manifested by adenoid facies.

X ray examination of the chest of asthmatics should not be omitted. Ordinarily the findings in the X ray plate of an asthmatic chest are useful only to rule out other diseases. In long-standing cases we often find considerable peribronchial infiltration with the characteristics of emphysema.

Laboratory studies of the blood, urine and sputum although not productive of findings characteristic of allergic disease should always be done. As with the X ray examination, they are important in ruling out other diseases. In passing I might mention that eosinophilia in allergy although frequent could scarcely be called characteristic or important in diagnosis.

Protein Skin Tests.

We now come to the final and perhaps the most important examination of the allergic individual, the protein skin tests, on which we must rely for obtaining the knowledge wherewith to administer specific treatment.

Two methods of performing the skin tests are in common use, the scratch method and the intradermal method. Briefly, the former consists of superficially scratching the skin to a depth insufficient to draw blood and on this scratch placing a bit of the testing substance, either in solution or by adding a solvent such as $^{n}/_{100}$ sodium hydrate solution to the dry substance already on the scratch. With the intradermal method, the substance already in solution is injected into the skin in just sufficient volume to raise a small wheal. By whichever method the test is performed, the reaction is interpreted in fifteen to thirty minutes. Grading of reactions for practical purposes is made on a basis of four degrees of severity. One indicates a doubtful reaction, usually merely an area of erythema surrounding the scratch with no wheal formation. This type of reaction in children may be considered definitely positive. Two is a definite but small wheal formation with pseudopodia surrounded by an area of erythema and accompanied by itching. Three is the same as two but larger and more intense and four is any characteristic reaction larger than 0·5 centimetre in diameter, appearing more rapidly than usual and accompanied by marked itching.

The factors modifying allergic (Larsen, Paddock and Alexander [17]) skin test reactions may be listed as depending on (i) the preparation used, (ii) the amount of protein brought into contact with cells and amount of solution injected, (iii) the method of application, (iv) the degree of cellular activity. These I will discuss in order.

The Preparation Used.

A first essential in protein skin testing is the use of test proteins known to be active. In my hands the commercial proteins, particularly the food proteins, have proved distinctly unreliable. Positive reactions obtained with these proteins can be accepted, but "negative reactions"[1] mean nothing and such negative reactions are so common that I am convinced that commercial food proteins are often physiologically inactive and therefore unsuitable for protein skin testing. This has led me to prepare all proteins in my own laboratory. In general the method is one of extraction of the well-macerated food substance with a buffered salt solution dissolving out the protein. To this solution acetone is added and the precipitated protein dried and ground to a palpable powder which is used for testing. The animal dander protein is prepared (Wodehouse[18]) by a process of extraction with an alkaline solution and precipitation at the iso-electric point. With the pollens the method (Clocke[19]) is that of a simple extraction in a glycerine and salt solution and of preparing a clear solution by filtering through a Berkefeld filter. The powders and solutions are tested clinically for their activity on known sensitive individuals. This procedure has resulted in the production of a group of some two hundred food and dander proteins and some one hundred and fifty pollen extracts of proven clinical activity and what is also of importance of almost unlimited stability in keeping quality.

The Amount of Protein Brought Into Contact With the Cells and Amount of Solution Injected.

Undoubtedly the size of the cutaneous reaction depends on the concentration of reacting substance brought into contact with the sensitive cells. To measure such concentration with the purpose of foretelling the activity of a given test protein is only roughly possible. My own experience (Piness, Miller and Alles[20]) has been that neither the total nitrogen content nor the protein nitrogen content can be used to foretell the activity of a protein.

The Method of Application.

As a routine I use both the scratch and intracutaneous methods of testing. Both are useful and yield excellent results, provided each is used in its proper place. It is my opinion that given an active protein and a sensitive individual positive reactions will be obtained. It is admitted that the intracutaneous method is more delicate and gives a greater number of positive reactions, but it has its disadvantages as well. It gives a greater number of pseudo-reactions, causes many constitutional reactions and may result in anaphylactic shock and death (Lamson[21]). On the other hand the scratch method has many advantages over the intracutaneous one. The technique is simpler, more rapid, less painful and less apt to produce constitutional reactions. Several constitutional reactions have been observed during the use of even this method, but not comparable in either number or severity with those observed by workers employing the intracutaneous method exclusively. The scratch method is the one of choice for testing children. Children on the whole react better than adults and a greater number of tests can be done at one sitting than by the intracutaneous method. One should not test to more than forty-eight proteins at one sitting, better half that number, particularly in markedly sensitive individuals, because of the danger of constitutional and local reactions. The average patient is tested with three hundred and fifty proteins. When doubtful or "negative" reactions are obtained in individuals who are either suspected or known to be definitely sensitive, I then resort to the intracutaneous method and these tests may be repeated several times until significant results are obtained.

The Degree of Cellular Activity.

The essential principle upon which the whole theory and practice of protein skin testing is based, is that the skin of the asthmatic individual exhibits a cellular activity similar in every way to that of the lining mucous membrane of the respiratory tract. Several factors modify this activity and must be borne in mind in performing, interpreting and evaluating the results of skin testing.

In performing skin tests one must realize that individuals vary in their sensitiveness to trauma. To avoid ambiguous results a control scratch must be made on which nothing is placed.

In interpreting reactions one should bear in mind that some proteins used for testing, notably egg-plant, spinach and blue-fish, contain substances which are irritating even to normal skins and frequently produce pseudo-reactions. Testing of all proteins on normal individuals obviates the need for any confusion which may arise from this source.

In evaluating reactions it must be remembered that the size of the reaction is not a measure of its ætiological importance, as we shall see in the discussion of treatment. Furthermore, the size of the reaction with the same test material on the same person at different times varies greatly. At times I have found it impossible to elicit a positive reaction either in an individual formerly giving strongly positive ones or in an individual in whom the history definitely pointed to a protein sensitization. I have also observed a group of individuals in whom definite positive reactions having been obtained and as a result of eliminating the substance from the diet over a period of from six months to several years the cutaneous reaction and symptoms disappeared. At the end of this time ingestion of the forbidden food caused a reappearance of both symptoms and cutaneous reactions. Schloss[22] has explained these phenomena as analagous to the phenomenon of anti-anaphylaxis in the experimental animal. The practical importance of this concept lies in its indication for the necessity of repeated testing in the individual who does not at first react.

[1] Dr. Piness uses this contradictory term which is in common use. Absence of reaction is meant. Editor.

TREATMENT.

It is my purpose under the heading of treatment to limit myself to a discussion of only that phase of treatment based on the results of the skin tests, that is specific treatment as opposed to symptomatic and general medical treatment whose importance I wish to emphasize in passing.

Specific treatment is based on the supposition that any particular protein which has given a positive reaction is ætiologically important providing that with it we can by inhalation, ingestion or subcutaneous injection reproduce the symptoms of allergy at will and that it is or has been present in the environment or diet of the individual (Cook[23]). To reproduce symptoms is not difficult; it is, however, dangerous and seldom resorted to. On the other hand to demonstrate the presence of the offending protein either in the diet or environment is of utmost importance. Particularly is this true of pollen proteins for to treat a patient with a pollen protein, not present in the environment, even though positive reacting, is useless and reprehensible (Piness[24]).

Pollens.

This necessity for determining the pollen environment of hay-fever and asthma sufferers has led to numerous extensive botanical surveys (Piness, Miller and McMinn[25]) of wind-pollinated plants and rightly so for the knowledge so obtained and correlated with the results of skin tests is the first step in successful treatment. This together with a history of the seasonal character of symptoms determines which pollens are to be made up into the antigen which is to be administered to a given patient.

Preseasonal treatment is the method of choice and should be commenced about fourteen weeks prior to the expected onset of symptoms and treatment given at intervals of about five to seven days dependent upon the time that the patient appears for treatment and the interval remaining before the expected onset of symptoms, decreasing the interval as the season approaches.

The initial dose is determined by the reactions obtained when the patient is tested to various dilutions of the antigen. The first dose is 0·1 cubic centimetre subcutaneously of the strongest dilution that fails to give a skin reaction. The dosage is increased at such rate as to complete the treatment prior to the date at which the onset of symptoms is expected, usually about fourteen weeks after beginning treatment.

Coseasonal treatment is sometimes necessary when a patient appears for the first time during the season of symptoms. However, in my hands and those of my coworkers it has been found most difficult and results are far from satisfactory when compared with the preseasonal method. The method employed by me is merely a modification of that described by others and is as follows. The initial dose, consisting of 0·1 cubic centimetre of the strongest dilution to which the patient failed to react, increasing 0·1 cubic centimetre each day, instead of every five to seven days, as described above, until the patient is free of symptoms, then increasing the interval between treatments, but continuing with the dosage which gave relief of symptoms, never increasing once a desensitizing does is determined.

With both methods local and general reactions must be guarded against. Local reactions consist of intense itching, redness and swelling at the site of injection. General reactions consist of the production of or the aggravation of the symptoms of hay-fever or asthma with at times a generalized urticaria within a few minutes to twenty-four hours after the injection. The occurrence of either a local or general reaction requires that the next injection consist of the same or smaller dose than that producing the reaction.

The results of treatment of pollen sensitive individuals have been very satisfactory, there being some degree of relief in more than 90% of the cases.

Foods.

Specific treatment of food sensitive patients is unsatisfactory and unnecessary in the great majority of cases, since satisfactory results can be obtained by elimination of the offending food proteins from the individual's diet. When such staple articles of diet as eggs, wheat and milk are the offending substances, their elimination is a serious matter requiring considerable ingenuity in the preparation of a satisfactory diet. For instance, sensitization to eggs means the elimination of all such dishes as custards, mayonnaise, pastries, ice-cream and so forth. Sensitization to wheat means the elimination of practically all breads, for nearly all breads rely on the gluten of wheat flour to hold the loaf together. In the same manner sensitization to milk, especially in the infant is with difficulty treated by elimination and such substitutes as goat's for cow's milk being made. Specific treatment against food hypersensitiveness has been attempted. Several authors have given gradually ascending doses by mouth of the offending protein until such time as the individual was able to tolerate large amounts. The second method has been that of giving the protein subcutaneously in gradually ascending doses. I have seldom seen satisfactory results obtained by these methods and therefore advocate the elimination of the food protein from the patient's diet over a period of years, depending upon the age of the patient and the results of semi-annual retesting. Children as a rule lose their sensitization to foods as they grow older and elimination during the sensitive period is the method of choice for the treatment of this particular group.

Epidermals.

Those individuals hypersensitive to the fine epithelial scales from animals such as horse, dog, cat, chicken, goose, rabbit, guinea pig, cow *et cetera* are best treated by avoidance of contact. This is not always possible because of environment or occupation, particularly in the case of individuals sensitive to horse, dog and cat. The technique of the treatment for this group is similar to that of pollen

therapy and the results are consistently good thus warranting one in treating this group when circumstances require.

House Dust.

House-dust sensitization has already been discussed. It can scarcely be considered under the head of specific treatment because of the vagueness of our knowledge concerning its constituents. My practice is never to make use of house dust in treatment until all other possibilities have been exhausted. The results of such treatment in the few cases in which I have employed it, have been far from satisfactory.

Bacteria.

In view of the fact that sensitization to bacteria cannot be demonstrated, treatment with bacterial vaccines is limited to those cases, usually perennial, asthmatic in type, in which no positive reactions could be obtained after repeated tests. Such cases are usually in elderly individuals who have either had asthma for many years or whose asthma began too late in life. The diagnosis here is usually that of asthmatic bronchitis and the use of either autogenous or stock vaccines has proved of equal benefit. Results are spectacular in a certain small percentage of individuals.

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DR. E. F. D'ATH (Dunedin) said that the pathologists had been making pollen protein in the Dunedin laboratory for twelve months and were carrying out the specific treatment of asthma. They had prepared a full series of pollen extracts and were beginning to prepare extracts of foods and of epidermis of various animals. He had followed Dr. Piness's methods very carefully. In regard to the treatment of hay fever they had obtained very good results from pollen extracts.

DR. J. HARDIE NEIL (Auckland) said that in examining noses of female patients he had often found white powder in the nostrils. These women used face powder. He thought that this powder must be a potent cause of asthma in allergic subjects. He complained of the uselessness of some of the commercial protein preparations on the market for the testing of allergy. He considered that the medical profession should be asked to support the laboratories connected with the public hospitals in the larger cities. If medical practitioners treated their allergic patients skilfully, a large amount of quack work would be eliminated. He was in favour of an acceptance of Dr. Piness's offer to train a graduate for six months at his laboratories.

DR. R. PULLEINE (Adelaide) raised the question whether asthma was of frequent occurrence in sinus disease. They knew that the removal of nasal obstruction brought about the disappearance of asthma in some patients.

DR. PINESS in reply said that he was sure that if they supported their local laboratories, they would obtain excellent material. If the University of Otago wished to send a graduate to his laboratories, he would welcome him and train him. Any practitioner would be welcomed at his laboratories. He had no secrets and would show visitors all his methods.

To Dr. Pulleine he said that he had had a series of eight hundred and thirty-four allergic patients who had had operations performed on their noses and throats. Not one had derived any permanent benefit. Nasal surgery would not cure the allergic condition. Polypi and obstructions were the result and not the cause of the allergy. The conditions should be treated independently. In children a

lymphœdema in the region of the posterior pharynx and a boggy condition of the nose were not signs of sinus disease. They indicated a potential allergy. It was useless to treat the sinuses in these children. The fact that they were well in the mountains and suffered when at home, was characteristic of allergy.

SECTION IX.—PÆDIATRICS.

ERYTHRŒDEMA OR SWIFT'S DISEASE.

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GREAT are the uses of publicity. This is well seen in relation to Dr. Swift's short paper on erythrœdema read at the Australasian Medical Congress held in Auckland in 1914.

The Transactions of this Congress were not issued until 1916 and it was not until then that Dr. Swift's paper was available to the medical profession. He sent copies of his paper to America, but heard no more of them. *The Lancet* was the only medical journal that gave a *résumé* of this paper in 1918 and Dr. Doak wrote to *The Lancet* in May, 1918, saying that he had seen at the Bradford Infant's Hospital several patients with a condition similar to that called erythrœdema by Dr. Swift. In 1920 many cases were reported and American writers began to contribute to the literature of this disease very freely. The first set of cases was collected by Dr. Patrick, of Portland, Oregon, who submitted the histories of eight cases to Dr. Weston and other pellagra experts, but their identity with this disease was denied and Dr. Sandwith, of London, in a letter to Dr. Weston, suggested their possible resemblance to acrodynia, the name given to an epidemic that occurred in France in 1830. Dr. Weston adopted this name acrodynia and redescribed Dr. Swift's disease in a paper which was shortly reported in the June number of the *Archives of Pediatrics,* 1920 and published in full in September of the same year. Dr. Byfield at the discussion on this paper said that he had already collected notes of seventeen cases of this same disease and he published a paper describing these cases in November, 1920, under the title "A Polyneuritic Syndrome Resembling Pellagra-Acrodynia, Seen in Very Young Children."

Since 1920 cases of Swift's disease have been reported from Great Britain, the Netherlands, Switzerland, Sweden, Poland, America, New Zealand and Australia. The disease is therefore present amongst children all over the world and as its symtoms become more generally known, the medical profession will be less puzzled as to the diagnosis.

The clinical entity of this disease is well recognized, now that we find the word erythrœdema in two of the latest text-books on diseases of children, namely, in the late Dr. John Thomson's "Clinical Study and Treatment of Sick Children" and in the latest edition of Garrod and Still's "Diseases of Children."

Dr. Swift is not satisfied with the name erythrœdema which he coined for his syndrome, as there is swelling, but no œdema of the hands and feet.

The name "pink disease" is not at all distinctive; Dr. Clubbe of Sydney, writing to me on April 19, 1923, stated: "I have always called these cases pink disease, but I do not know if I originated the name."

Acrodynia is a wrong name to use. It is already the name of a disease which apparently was an arsenical peripheral neuritis with painful extremities going on to paralysis and atrophy. These cases occurred in epidemic form in France in 1830 amongst adults, chiefly in asylums and was probably due to arsenic getting into the wine. A similar epidemic occurred amongst beer drinkers in England in 1900. As we have no knowledge up to the present time of the ætiology of the disease, it would be a courteous act to follow Dr. Warthin, of the Pathological Laboratory of the University of Michigan, who suggests that we should call the disease Swift's disease. A sub-leader in *The Journal of the American Medical Association* of April 10, 1926, cordially endorses Dr. Warthin's suggestion. Dr. Swift's name should go down to posterity like the names of Barlow, Bright, Graves, Pott and Colles.

Although articles innumerable have been written since Dr. Swift's paper, practically no new symptom has been described and nothing of value has been added as to the ætiology of this strange affection that seems to be confined to infants and young children.

Since reading my paper at the Australasian Medical Congress in Brisbane in 1920 I have seen a second series of forty-three cases, twenty-one being in males and twenty-two being in females. The youngest patient in this series was five months and the oldest three years and six months. In 1916 I began to diagnose these cases under the heading of erythrœdema and I have not seen a patient under four months of age or one older than three years and six months. Weston, of Philadelphia, reports a well-marked case in a boy of five years and McKenzie, of Canada, also had a case well marked at five years of age. The greatest number of cases occurs between nine and eighteen months. Twenty-six of my last forty-three cases occurred between these ages and nine below nine months and only eight cases after eighteen months.

Out of 131 patients reviewed in this and my first paper 83, namely over 63%, have been between the nine and eighteen group. Practically all the infants and children who suffer from this complaint, have had excellent health up to the time of onset of the first symptoms of Swift's disease. They have been healthy strong infants, nearly always breast fed and showing excellent weekly gains in weight up to the time they have gone off their food.

To give two instances.

F. McN., a male, *ætatis* nine months, was seen on August 14, 1925. He is the fourth child; the others are all well. This child was on the breast which he takes every three

hours; his birth weight was four and a half kilograms (ten pounds). He was quite well up to seven months; he then weighed eleven and a third kilograms (twenty-five pounds) and was beginning to crawl, when a girl charged into the perambulator in which he was lying and shook him up and made him cry. He slept badly that night and since then has had wretched nights. On August 13 he cried from 10 p.m. to 3.45 a.m., he then slept fitfully to 6.30 a.m. For eight weeks he had not had more than three or four hours' sleep at night; he was sweating freely, had a rash all over his body which was terribly irritable and bit at his mother's arms; his muscles were wasted and limp; his hands and feet were icy cold, red and swollen, his mouth had been ulcerated, but was better. His weight was nine and a half kilograms (twenty-nine pounds), that is he had lost 1·8 kilograms in eight weeks. He slept with his head bent down between his knees. He was kept in the open air day and night, rubbed with methylated spirit and dressed in Fuji silk and covered very lightly. He was also given condensed milk to supplement the breast, as much as he would take, as well as fruit juice.

He slept well later up to September 7, 1925, when he again began to sweat freely, "steam arising from his body and head." His temperature on September 21 was 39·5°C. (103·2°F.). There were dulness and increased vocal resonance at the base of the left lung posteriorly. He had a croupous pneumonia; the crisis occurred in seven days and he made a complete recovery. The duration of the illness was four months.

W. T. H., a male, *ætatis* eleven months, was seen on August 12, 1924. He had been fed at the breast up to three weeks before, when "he was weaned because he was suffering from teething." He sat up at six months and was crawling and pulling himself up and standing with support at nine and a half months (that is six weeks before date of visit). He then went off his food, had a rash over his body with no sore throat and a temperature of 39·4°C. (103°F.). His temperature was raised for about four days and his rash disappeared in a few days. Subsequently he did not sleep at night and lost his appetite for food. He was sweating freely over the body, but no rash was present. His hands and feet were red, swollen, moist and icy cold. No catarrhal symptoms were present with the fever. He was put to bed at 8 p.m. on August 11, awakened at 8.45 p.m. and stayed awake until 9.30 p.m., then slept till midnight, when he awakened and slept again to 2 a.m.; he then cried and grizzled until 11 a.m. next morning when I saw him. The pronounced sweating of the skin had been present for at least five weeks. The only sign of a rash on the body was a slight rash round the region of body where the elastic band of the modisties was situated. His birth weight was 3·6 kilograms (eight pounds). It had been up to nine kilograms six weeks previously. At the time of the visit it was seven and a half kilograms (sixteen pounds, eight ounces).

On August 22, 1924, he was sweating very freely. His clothes had to be changed four times during one morning. After an afternoon on the beach he slept from 9 p.m. to 2 a.m. He then tossed about until daylight. He was dressed in silk. He was drinking orange and passion fruit juice freely and a quart of raw milk a day. His bowels were opened with castor oil every second day. He had ulceration at the back of the upper incisor teeth where the lower incisors seem to strike the gum. His rectal temperature was 38·5°C. (101·4°F.). His weight was 7·2 kilograms (fifteen pounds, fourteen ounces).

On September 2, 1924, he was still sweating very freely; he slept better for a few nights and had again been getting restless during three nights. He was still taking a quart of milk a day and was taking a little bread and butter. His bowels were open naturally twice a day; the motions were healthy. He was drinking water freely. His rectal temperature was 37·6°C. (99·8°F.). The gums had improved after he had been allowed to chew at a piece of absorbent wool wet with peroxide of hydrogen solution. His weight was 7·8 kilograms (sixteen pounds, five ounces). On November 27, 1924, he had been sleeping well for four weeks. His weight was 8·85 kilograms (nineteen and a half pounds). His hands and feet were still reddened, but there had been no sweating for the past month and he was very bright and well. The duration of his illness was about four months.

On what diet are these children when the early symptoms first appear? I have investigated fifty consecutive cases and find that thirty of the children were on the breast when the symptoms commenced, four had been weaned and bottle fed under four weeks of age. Nine children who showed symptoms between twelve months and three years, had all been breast fed up to eight or twelve months or more. The mothers nursing these children had lived under good circumstances in a country where fresh food is plentiful and so the question of avitaminosis cannot come into the question of ætiology.

I have seen two cases occur in one family. A mother of one of my early patients, having had four months of worry and anxiety and sleepless nights, asked me if there would be any chance of a second child getting erythrœdema if she should have another child. I told her that up to that time I had never heard of two cases occurring in one family. Two years later she brought me her second child with definite symptoms. Both children recovered well.

With regard to infectivity, one mother I saw this year tells me that she called at a house near Melbourne where the baby was suffering from pink disease. Her child who accompanied her was in the same room as the affected child and a month later her own child began to be fretful and irritable and was brought to me later with definite symptoms of Swift's disease. A number of writers mention severe colds or bronchitis as early symptoms marking the invasion of the disease. But most mothers say that the illness began by the children refusing to take the breast well, loss of weight and constant fretfulness day and night. These symptoms preceded the sweating of the skin as a rule. The sweating of the skin is usually well developed before the miliarial rash appears and the redness and coldness of the hands may not develope for eight to twelve weeks after the commencement of the affection. I have seen one patient with pronounced signs which got better in three months without any redness or peeling of either hands or feet.

The stomatitis may be an early symptom. I saw one child in my consulting room on November 10, 1926, a girl aged one year and nine months, with a history that she had had a sore mouth for ten days. She had an ulcerative stomatitis with a temperature of 38·6°C. (101·6°F.) in the rectum. I also made a note to the effect that she had been very restless for five weeks and that she had been off her food. I received a shock when I saw the child again on December 3, 1926. Directly she came into the room the diagnosis of Swift's disease was evident by the whining, bent-up, fretful child with a red peeling hand lying limp by the child's side. In the interval between the two visits the skin had commenced to sweat freely and the hands had become red and the skin had begun to exfoliate. In this case the hands became affected about the sixth week of her fretfulness. In other cases the stomatitis has followed on

a pustular affection of the fingers and as all these children are prone to suck their fingers, I had for a time not only tried to keep the fingers sterile with tincture of iodine and methylated spirit, but had also splinted the elbows to prevent mouth infection. I have, however, been forced to the conclusion that the stomatitis is frequently present when the fingers and hands are unaffected. I do not know if the type of the affection is milder now than previously, but have not seen the severe ulceration of the gums since sterilizing the hands daily, that I saw amongst the patients eight and ten years ago. One child recently at the Children's Hospital chewed at her lower lip to such an extent that when she died, it was in a bad sloughing condition.

The excessive sweating is a most troublesome symptom and necessitates the frequent changing of the sodden underclothing. It may be most evident at one time in the day necessitating changing the clothes four times during twelve hours; at another time you can detect a slightly moistened skin only when the hand is inserted under the clothes. Then again without apparent reason the drenching sweating reappears. It is one of those symptoms that flatters you that you have done some good with treatment, only to disappoint you again in a few hours or days.

The rash seems to bear a distinct relation to the sweating and nurses tell me that the sweating and rash come and go several times during the twenty-four hours, the rash being most pronounced when the sweating is profuse and then when the skin is dry, the rash may be barely perceptible.

In my second series I have not seen the severe ulceration about the fingers and buttocks that I saw in my early cases and feel certain that the sterilization of the skin with iodine and methylated spirit has gone a long way in preventing ulceration and furunculosis so common in macerated skin conditions.

Dr. Giblin wrote to me from Papua on June 12, 1921, to tell me of a case he diagnosed at Samarai in which there was suppurative arthritis of the metacarpo-phalangeal joint of the first left finger, also of the first interphalangeal joint of the right second and third fingers. These joints were all incised and creamy pus evacuated. The joints healed quickly within a week. This child's lower incisor teeth dropped out and necrosis of the lower jaw occurred. This is the only case of infection of the joints of hand that has been reported.

Photophobia has not been such a prominent symptom in my last series. When it is present there is usually a conjunctivitis without secretion. It, like the sweating, has an irritating way of recurring several times after apparent subsidence. Children sometimes lie for weeks with eyes tightly closed.

To see if I could find any seasonal occurrence of these cases I took thirty-one successive cases to see what month in the year the symptoms began and found that from January 1 to June 3 four patients had their first symptoms, but from July 1 to December 31 the illness of twenty-seven patients began. In eighteen of the thirty-one patients it began during our winter months and the other thirteen in the warmer months of the year. So I am afraid we cannot look for a seasonal cause in the ætiology of this disease. Dr. Littlejohn's experience in Sydney (Australia) was that his cases usually were seen in the late summer and autumn months coincidently with cases of infantile paralysis.

Prognosis.

Since 1920 I have had to qualify my opinion as to the prognosis in these cases and to say that in the open air and better still out of the city the prognosis is good as to ultimate recovery, but when it is necessary to send these patients into the wards of a general or children's hospital the mortality amongst them is very great. During the past six years thirty-one patients with Swift's disease were admitted to the Melbourne Children's Hospital and no less than ten of these children died. When children suffer from this disease, their resisting power against infections of various kinds is considerably lowered and broncho-pneumonia and colitis are frequent causes of death. One of these hospital patients died suddenly in bed without showing any very definite pathological reason for her death. The night nurse reported that she had not slept at all for some nights, either lying quiet or tossing about. Also one of my own patients in private seemed to be doing well under ultra-violet ray treatment and her mother was hoping for a gain of a few ounces for the week, but she died quietly in her sleep most unexpectedly. This sudden death has now occurred twice amongst my patients, once in Dr. Swift's experience and once at the Children's Hospital, Melbourne.

With regard to the deaths at the hospital two children died within the twenty-four hours of admission and five others within fourteen days, showing that the children were seriously ill on admission, which accounts to some extent for this high mortality.

Pathology.

Regarding the *post mortem* appearances I have nothing new to publish in this connexion. The best contribution I have read so far is a paper by Dr. Aldred Scott Warthin, of Ann Arbor from the Pathological Laboratory, University of Michigan, United States of America. He reports the *post mortem* appearances in two children who died in the University Hospital of Michigan. The histories given were very typical of the disease and he sums up his findings as follows:

"Acute infection of the respiratory tract was present in both cases; in one case an older process. Both cases showed gastro-enteritis. No other infective processes were present. Both showed marked features of the hypoplastic lymphatic constitution. In both cases there were extreme congestion and œdema of the central nervous system, with reticulo-endothelial proliferation. Both cases showed inanition in the serous atrophy of the adipose tissues.

The essential pathological changes in these two cases would appear to be extreme œdema and slight

meningeal irritation of the central nervous system, chronic erythema of the skin with hyperkeratosis, hypertrophy of the epidermis and sweat glands, with slight pigmentation of the rete, occurring in children of the hypoplastic lymphatic constitution, with associated or terminal respiratory infections and gastro-intestinal catarrh and inanition.

These two cases, therefore, do not support the theory of a polyneuritis as the essential feature of the disease; neither do they justify the use of the term of erythrœdema as a fitting designation, as they manifested no œdema of the corium. They do not present any evidence as to a specific infectious ætiology; they likewise show no relationship between tonsillar disease and the syndrome.

The changes in the skin and central nervous system suggested to me at once the pathology of the early erythema stage of pellagra. The changes in the skin are also identical with those seen in certain forms of light sensitization, xeroderma and fagopyrism. They are also identical with certain stages of Röntgen ray and ultra-violet ray erythema. The entire anatomical picture in these two cases suggests either a food deficiency or a toxin acting on persons of the hypoplastic constitution, affecting the reticulo-endothelial system of meninges and skin, the vegetative nervous system and possibly leading to a light sensitization. May it not be as was early suggested by Byfield, a condition closely related to pellagra, perhaps an infantile variety of that affection? Other necropsy cases must be collected and studied before this question can be settled. In the meantime it would be better to call the condition Swift's disease than to designate it by terms that apply so little to it, as do 'acrodynia' or 'erythrœdema'."

Treatment.

The past six years has not brought forward any form of treatment that will reduce the duration of the illness to less than three months' suffering. Dr. Bruton Sweet's suggestion of the good effect produced by ultra-violet ray treatment was only brought under my notice about three months ago and I immediately tried it, but as already reported, after apparently sleeping better after three exposures to the lamp, the child died suddenly in her sleep.

This treatment was administered in the skin department of the Children's Hospital to a patient who subsequently died in the hospital from bronchopneumonia after she had chewed her lower lip into a badly sloughing condition. These children are most comfortable if allowed to lie practically unclothed. Exposure of the naked skin to the direct rays of the sun is most irritating to them, the heat rays in the sunlight apparently causing intolerable irritation. This made me feel very hopeful regarding the ultra-violet ray lamp, which is sunshine without heat. If any clothing is put on these children, it must be of the lightest description and also of a material that absorbs moisture quickly. For this reason cotton or silk is preferable to wool and infinitely more comfortable. Bed clothing should be used as little as possible. They sleep better without it. The skin should be kept as free from streptococcal and staphylococcal infections as possible and with this object in view I do not know of any better way of securing this than daily rubbing of the skin of the whole body with methylated spirit and dusting with a drying powder. The hands are kept free from ulceration and onychia by painting with iodine tincture weakened to half strength with methylated spirit.

The hands and feet are not tender and painful as is suggested by the term acrodynia, the patient seems to get relief by constantly scratching at them or when the mother is rubbing them. The irritability of the hands and feet is certainly increased by hot bottles and blankets and relieved by exposing them to cold air.

With regard to the stomatitis the gums and inside of mouth should be swabbed with absorbent wool soaked in peroxide of hydrogen solution one part, water four parts, followed by "Listerine" one part in eight. If any ulcers are obstinate in healing, they may be touched with 1% chromic acid solution. In bad cases of ulceration and sloughing of mucous membrane of the mouth and lips, the affected parts should be douched with peroxide (one part in four of water) followed by normal saline solution.

Fresh air day and night, fresh milk, fresh fruit and practically no clothes and no attempt to amuse the children will see most children get well in three months or longer.

We still want something to make these children sleep well at night and also some treatment that will be sure to terminate the symptoms in the first month, instead of waiting three months to see them recover.

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THE TREATMENT OF ERYTHRŒDEMA.

By G. BRUTON SWEET, M.B., C.M.
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In the *Archives of Pediatrics* of August, 1925, an article was published by myself dealing with the treatment of erythrœdema and detailing five cases, three of which were treated with satisfactory results with artificial sunlight.

Since then four further cases have been met and all were treated in a similar manner with an equally happy result. Brief particulars of the histories of three of these patients will appear in this paper; the other is mentioned in somewhat greater length on account of an unusual feature, namely the late appearance of the well-known cutaneous signs and also on account of the severity of the case.

Although the number of cases, seven in all, is small, the results have been so uniformly satisfactory that I have come to the conclusion that artificial sunlight is a specific for this otherwise obstinate and troublesome condition. It must be admitted that up to the present no other observer has reported similar results; indeed the only reference that I have been able to discover is a paper on erythrœdema by Lapage published in *The Lancet* of January 23, 1926, in which the treatment of ultraviolet rays is mentioned only to damn it with faint praise.

In England and America this disease appears to be rare; at any rate the published cases are few in number. In Australia on the other hand judging by the reports of Swift, Wood and others, it is by no means uncommon and it is in the country where it was first discovered that we should look for a cure. It will be interesting, therefore, to ascertain to what extent this treatment has been tried in Australia and whether similar happy results have been obtained.

When in Sydney a few months ago I was informed that artificial sun treatment had only recently been used for erythrœdema at the Royal Alexandra Hospital for Children in Sydney, but with satisfactory results. Most of the authorities on this disease express the opinion that the ultimate prognosis of erythrœdema is good and that though the duration of this disease is a lengthy one, the great majority makes a complete recovery. An analysis of the records of patients admitted to the Royal Alexandra Hospital on account of this disease does not quite bear this out.

During the years 1924 and 1925 twenty-six patients were treated and of these only three were said to have been cured, twelve were relieved, one unrelieved, seven died and three remained under treatment. A percentage cure of only 11%, with a case mortality of 27%, leaves much to be desired and indeed the Medical Superintendent of this institution informed me that in connexion with this disease it was customary to give a very guarded prognosis to the parents. The figures quoted above are striking in contrast with the result of my last seven cases with 100% of cures. The following are summaries of the histories of patients seen by me which have not yet been reported.

CASE VI.—M., aged four and a half months, was first seen on September 15, 1925. She cried night and day and had done so for the last three or four weeks. She was getting worse and losing weight; she vomited a little and was very constipated. She had been breast-fed entirely until a month previously. At the time of examination she was partly on the breast and partly on humanized milk.

Her nutrition was moderate. Her weight was 5·9 kilograms (thirteen pounds). She had a typical raw beef appearance of her hands and feet and a fine papular rash all over her body.

Treatment with artificial sunlight was commenced on September 25, ten exposures were given at three of four days' interval. On October 3 there was a definite improvement as regards sleeping and general happiness. The improvement after that was continuous. The last treatment was on October 28 when all symptoms had disappeared, but her weight was only 5·5 kilograms (twelve pounds two ounces). After this her weight gradually increased and when seen for the last time on March 1, 1926, she was a perfectly healthy baby weighing 7·15 kilograms (fifteen pounds twelve ounces).

CASE VII.—D., aged eleven months and three weeks, was first seen on December 7, 1925. He had been ill for a month. He was losing weight, sleeping little night or day. He sweated a great deal, was constipated until shortly before admission, when his bowels became loose. He had been breast-fed for only one month and after that humanized milk and latterly "Neave's Food" in addition had been given.

His nutrition was moderate; his weight was 8·28 kilograms (eighteen pounds four ounces). There was a typical appearance of his hands and feet and a fine papular rash on body; he had swollen and tender gums.

Treatment with artificial sunlight commenced on the same day. Improvement was very rapid. He slept well after two exposures. The sweating and rash in this baby were the last signs to disappear and this was probably due to over-clothing during summer weather. This baby gained weight steadily during treatment, in this way differing from nearly all the others so treated. Nine exposures were given with the mercury vapour and carbon filament lamps and recovery was total and complete and on January 21 he weighed 9·5 kilograms (twenty-one pounds).

CASE VIII.—J., aged fifteen months, was first seen on February 13, 1926. She had been ill for about three months or longer. She was extremely irritable, slept very little night or day and was constantly crying. She sweated a great deal. She took little food. Her bowels acted daily and the motions were loose and offensive. She was very wasted, with an expression of abject misery. There was a fine papular rash on the trunk and limbs. Her hands and feet had a normal appearance. Owing to the absence of redness of the hands and feet, the usual treatment was not recommended until the diagnosis was more assured.

On the following day the mother who was suffering from nervous exhaustion, due to worry and loss of sleep, called and begged me to get the child admitted to a hospital and an order was given and the patient was lost sight of for over two months when on my return from Australia she was discovered in my ward at the hospital. She had been for some weeks at the Auckland Karitane Home and had steadily got worse and had been an inmate of the Auckland Hospital for about six weeks. Her appearance was

pathetic in the extreme. Although seventeen months old she weighed less than six kilograms. She had developed typical raw beef hands and feet, with desquamation of skin and superficial ulcerations. There was pronounced intertrigo and large superficial ulcerations on both buttocks. The body was covered with marks of scratching. Rhinorrhœa and troublesome cough were present. Photophobia was present but no stomatitis.

Her parents had been informed that there was little, if any, hope of recovery. She was shown at the Clinical Society and a hopeful prognosis given.

Treatment with artificial sunlight was commenced on April 24 and she made steady progress. She had ten exposures and she gained weight during treatment. When last seen on October 11 she weighed 8·6 kilograms (nineteen pounds) and was a healthy happy child, but undersized for age.

CASE IX.— J., aged ten months, was first seen on August 21, 1926. She had been ill for two weeks. She had fever, cough and a rash on her face. She had been getting thinner and was irritable and did not sleep during the day, but slept fairly well at night. She had been breastfed for two months and since had been on humanized milk.

Her nutrition was moderate. Her weight was 7·8 kilograms (seventeen pounds four ounces). She had a typical appearance of her hands and feet. There were abrasions from rubbing on both feet; a fine papular rash on abdomen and chest was noted. There was erythrœdema of the thighs and buttocks.

Treatment with artificial sunlight was commenced on August 24 and eleven exposures given; all symptoms disappeared. She weighed less at the end of the treatment than at commencement. After that her weight gradually increased and on November 29 weighed 8·85 kilograms (nineteen pounds eight ounces).

Note on Treatment.

In all cases a combination of the ultra-violet rays of the mercury vapour lamp and the luminous and infra-red rays of the carbon filament was given. Whether the results obtained were due wholly to the former, the latter or the combination of these three light waves, I am unable to say, but I strongly advise for the comfort and well-being of the infant under treatment that at any rate during the winter months of the year treatment with the mercury vapour lamp should always be combined with radiant heat.

DR. E. H. M STEPHEN (Sydney) mentioned the case of a patient with erythrœdema whom he had treated successfully with pertussis vaccine. He had tried coryza vaccines for other children, but with no effect. Many of his patients with erythrœdema had had coryza. In speaking of the pathology of the disease he stated that there were no typical changes of the nervous tissue by which the disease could be recognized. In the treatment he had found parathyreoid glands of no value, but had had good results with ultra-violet rays. In one patient who had had rickets complicating erythrœdema, the restless movements soon disappeared after exposure to ultra-violet rays. He saw no reason why the child could not remain at home while being treated. He had frequently seen sudden death while the children were asleep.

DR. H. GRAHAM ROBERTSON (Wellington) said that treatment by ultra-violet light was successful for four reasons. It produced systemic uplift; it increased the calcium, phosphorus and iron content of the blood; it increased the hæmo-bactericidal power and it increased endocrine activity.

DR. A. PATTERSON (Wellington) spoke of the illness of a child in whom there was curious persistence of pustulation. After six injections of 1·5 cubic centimetres of colloidal manganese the pustulation had cleared up.

DR. A. JEFFERIS TURNER (Brisbane) said that the disease was a symptom complex. There were five symptoms. The first three symptoms were present in nearly every patient. The symptoms were anorexia and wasting, debility and sleeplessness, sweating and skin rash with redness of the hands and feet, photophobia and stomatitis. He mentioned two patients in one of whom there had been persistent photophobia, but no pathological change had been observed in the eyes. It was not until later that the typical symptoms of erythrœdema developed. In another patient the condition had not been diagnosed at the time; it was thought that it was a disturbance of nutrition. The patient had slept badly, but had none of the typical symptoms. There was no skin rash. In some patients there was no skin rash. Stomatitis had appeared and the patient had lost some teeth and a piece of the jawbone had necrosed. Turning to the question of prognosis, Dr. Turner stated that he always gave a favourable one. He thought that the reason that he had had no deaths was because he did not send his patients to hospital. In his treatment he regarded two things as essential, the keeping up of the child's weight and the giving of chloral and bromides or opium if necessary to soothe the child. He was not hopeful that a specific cure had been found. He was not convinced by the records of cures published. He thought that the patients got well of their own accord. At first he had regarded it as a nutritional disease, but he had changed his mind. It was not due to septic foci. He was inclined to believe that it was due to a virus of some sort affecting the sensory nervous system. He had not seen two members in one family affected.

Dr. Jeffreys Wood said that he always gave a good prognosis, that the child would be ill about three months. He warned them against hospitals because of the risk of infection. He always pointed out that there was a remote chance of sudden death.

Dr. Sweet agreed that it was better for the patients not to be admitted to hospital. In reply to Dr. Robertson he stated that he did not give calcium in treatment. His patients got rapidly better by treatment with ulra-violet light. In his opinion that treatment was quicker than any other known to him. He also mentioned a case of a patient in whom photophobia had been the first symptom and an eye specialist had been consulted. Nothing had been found amiss with the eyes; the typical symptoms had appeared later.

ULTRA-VIOLET LIGHT AND CHILDREN.

By H. GRAHAM ROBERTSON, C.B.E., F.R.C.S.E.,
Wellington, New Zealand.

PRESENT indications are beginning to show that actino-therapy is fast coming to be recognized as a settled branch of modern medicine with more and more understanding of its scope and possibilities. It must not be forgotten that it replaces nothing and is in most cases an adjunct only.

I do not intend to enter into an exhaustive analysis of ultra-violet light and its component wave lengths; but shall merely indicate briefly its place in the spectrum and its principal properties.

The ultra-violet light region extends approximately between 3,800 and 2,100 Ångström units and can be divided into a near, middle and far field.

Of these the near field between 3,800 and 2,960 is biotic, of no great germicidal action, but of greater penetrability. Such a region is available in any mercury vapour or carbon arc lamp and is most suitable for biological or systemic treatment. The better type of mercury vapour lamp emits light with approximately 28% of ultra-violet light and some 52% infra-red and other rays.

The region between 2,960 - 2,100, the abiotic region, has considerable germicidal action, but low penetrability; that region between 2,800 - 2,100 is claimed to have the maximum of bactericidal efficiency.

It is from the water-cooled mercury vapour lamp, free from radiant heat that the most suitable supply of abiotic waves are derived; though by the use of the ordinary mercury vapour lamp at short range, the action of the shorter rays can be secured before they are absorbed by the atmosphere. One cannot, of course, secure the benefit of lens pressure in any save the water-cooled type.

From the carbon or carbon impregnated arc lamps, one secures a light, containing some 5% ultra-violet light and 85% of infra-red, eminently suitable for institutional work, particularly for children and for sanatoria. It is primarily biotic and stimulating.

Other open arc lamps of richer ultra-violet ray content, up to 28% are available, for example, tungsten lamps.

The use and utility of ultra-violet light are bound up in varying combinations with its own factors and with the different wave lengths that represent luminous light, radiant heat, X ray, diathermy and so forth.

Instances of this are shown in the prior use of ultra-violet light before X ray exposures, increasing its effectiveness and lessening chances of X ray burns, for which by the way ultra-violet light is the best treatment. Also the combination of ultra-violet light and infra-red rays will restore more rapidly the musculature in tuberculous bone and joint disease and also in infantile palsy.

Again, *plus* diathermy, ultra-violet light is valuable in chronic inflammatory non-tuberculous processes, for example perigastritis, colitis, cholecystitis, pleuritis, chronic arthritis *et cetera*.

Let me briefly sum up the principal activities of ultra-violet light, the natural stimulant of the great metabolic organ formed by the living cells of the epidermis, the factor in the sun's rays that keeps the earth sweet and wholesome.

Firstly, it greatly increases the general metabolism, producing a definite "systemic uplift." It increases the hæmobactericidal action, due probably to an increase of antigens with consequent improvement of the defensive and resisting power of the individual. Its local reaction means erythema with its consequent blood changes and a definite leucocytosis follows general use. There is increased endocrine activity, especially parathyreoid. This appears to be the result of activating the hormones in the blood, with consequent stimulation of gland activity and coincident regulation of the balance of the whole endocrine system. It is possible that its action on the thyreoid, a key gland, may account for the major portion of this effect, especially in children, adolescents and those past middle life.

Again, we have increased vitamin activity, especially of fat soluble *A*; it is in this regard that the cure of rickets has been brought about largely, in addition to cod liver oil, calcium and so forth. It is definitely lethal to bacteria, their products and secretion, though our old friend the specific spirochæte is immune.

It definitely increases the calcium, phosphorus and iron content of the blood and as you will remember the calcium decrease in all exhausting fevers and tumour growths, you can understand the importance of this action.

A phase of the use of ultra-violet light that must be remembered, is the need for the closest collaboration with the attendant physician or surgeon. This must be thorough and mutual, as the coincident administration of drugs, vaccines, sera and other remedies is attended with risks that must be guarded against. For example iodism can be readily precipitated even in small doses of the drug.

The absorption and cell fixation of iron phosphorus and calcium is increased and favourably influenced by the use of ultra-violet light and should be helped by the administration of their salts. One finds often that relatively small doses will produce saturation point and one must watch for this accordingly or excessive excretion may result.

So with vaccines, as the negative phase can be intensified by ultra-violet light, care is necessary.

As any local intensive dose of ultra-violet light is followed by local, focal and general reactions, as in vaccine therapy, repeat doses must be timed accordingly. These reactions, of course, differ in their manifestations according to the disease, its nature, stage and severity, as also to the dose and source employed.

So with colloidal preparations and endocrines; so that we can understand the need for thorough collaboration between the physician and the light therapist.

Returning to the action of ultra-violet light in regard to calcium we can see what a field of usefulness is opened up in but one direction, namely the value of a course of general irradiation prior to all operations where hæmorrhage is feared. The increased hæmo-calcium content means largely increased blood coagulability, with a great "systemic uplift" in addition, whose value to the patient may be inestimable. This together with increased hæmobactericidal activity and leucocytosis means a great deal to the progress of your patient. So also its postoperative and postfebrile use has a value of no mean extent, as has been repeatedly proved.

Its local use covers an extent of ground impossible to traverse thoroughly in the scope of a paper such as this. One can but state that it is counterirritant, stimulating, bactericidal, analgesic, protein shock producing and of more than passing value in "regional" work through its reflex action on distant viscera.

The dermatologist could not, I think, do without its various activities covering from acne to xerodermia, from alopecia to anal pruritus.

The above summary indicates the basis on which the actino-therapist builds and indicates the wide range of physical and mental ills that constitute his field of activity.

(To be continued.)

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Wholly Set Up, Published and Printed at The Printing House, Seamer Street, Glebe, Sydney, N.S.W., by HENRY WILLIAM ARMIT, of The Point Road, Woolwich, Sydney, on behalf of THE AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED, of 30-34, Elizabeth Street, Sydney.